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CLINICAL ANALYSIS OF

NORMAL TENSION GLAUCOMA

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CERTIFICATE

This is to certify that the dissertation entitled, "**CLINICAL ANALYSIS OF NORMAL TENSION GLAUCOMA"** submitted by **Dr.V.DEVI PRIYA,** in partial fulfillment for the award of the degree of Master of Surgery in Ophthalmology by The Tamilnadu Dr.M.G.R.Medical University, Chennai is a bonafide record of the work done by her in the Regional Institute of Ophthalmology, Government Ophthalmic Hospital, Egmore, Chennai, during the academic year 2007 – 2010.

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INTRODUCTION

Glaucoma is one of the leading causes of irreversible blindness throughout the world and its clinical evaluation, early diagnosis and treatment remains a challenge to the ophthalmologist even today. World Health Organisation statistics indicate that glaucoma accounts for blindness in 5.1 million persons or 13.5% of global blindness.[1]

Traditionally glaucoma has been classified as primary and secondary forms. Within this large group of glaucoma, the most common form is primary open angle glaucoma, characterized by intraocular pressure > 21mm of Hg in atleast one eye, open and normal appearing anterior chamber angle and typical glaucomatous visual field loss or optic nerve head damage.[2]

At the other end of the spectrum, with regard to susceptibility to IOP are those patients with open, normal appearing anterior chamber angles, who have glaucomatous optic nerve head and visual field damage despite intraocular pressure< 21 mm of Hg on all occasions. They also have progressive glaucomatous damage with the absence of secondary causes for disc damage.

These patients are said to have normal tension glaucoma/ low tension glaucoma. Some investigators feel that normal tension glaucoma is a variant of Chronic open angle glaucoma. Normal tension glaucoma is defined as typical Glaucomatous optic disc cupping and visual field loss in eyes with normal IOP, open angles and absence of any contributing ocular specific systemic disorders. Normal Tension Glaucoma accounts for 30% of all glaucomas.[1]

HISTORICAL REVIEW

In Hippocratic writings the term “glaucosis” refers to bluish-green hue of the affected eye. This term included a larger group of blinding disorders like cataract . It was not until the nineteenth century that glaucoma was recognized as a distinct group of ocular disorders.

Von Graefe in the year 1857 first recognized optic nerve head abnormality with disturbance of vision & digitally normal tension.

In 1976, J. LAWTON SMITH suggested the theory of glaucomatous disc changes and field changes in an eye with a tension of 21 mm Hg. He had also formulated the possible etiologies for normal tension glaucoma.[1]

ANATOMY OF ANGLE OF ANTERIOR CHAMBER

The angle is bounded at anterior side by the peripheral part of cornea, the trabecular meshwork, the anterior face of the ciliary body and posterior wall is formed by the iris. The sclera groove lies between the scleral spur posteriorly and anterior border ring of Schwalbe's line anteriorly which is occupied by the canal of Schlemm and trabecular meshwork.

TRABECULAR MESHWORK

It is a triangular structure the apex of which blends with the termination of descemet's membrane and deep corneal lamellae. The base of the triangle is attached to the anterior surface of sclera spur, anterior surface of ciliary body and root of the iris. The sclera sulcus is converted into a circular channel called schlemm's canal by the trabecular meshwork.

Histologically it is composed of lamellae made up a central core consisting of ground substance collagenous and elastic like fibres, surrounded by a single layer endothelial lining that is supported by a basement membrane. The anterior part of meshwork is non-filtering and posterior filtering part is divided into 3 portions.

1. UVEAL MESHWORK :

This portion adjacent to the anterior chamber are arranged in bands that extend form the iris root and ciliary body and extend to the peripheral cornea. It contains irregular openings ranging in size from 25-75 microns.

2. CORNEO-SCLERAL MESHWORK

This portion extends from the sclera spur to the lateral wall of sclera sulcus. The openings varying from 5- 50 microns become progressively smaller as the schlemm's canal is approached.

3. JUXTA-CANALICULAR TISSUE

It is a thin layer of tissue 2-20 microns thick, the outermost portion of the meshwork adjacent to the Schlemm's canal.

AQUEOUS VEINS

They are exit channels of aqueous first described by Ascher. They vary in size from 0.01-0.1 mm and interconnect Schlemm's canal and episcleral veins.[1,4]

ANATOMY OF THE OPTIC NERVE HEAD:

Optic nerve head is defined as the distal portion of the optic nerve that is directly susceptible to Intra- ocular pressure.Optic nerve head is

composed of the nerve fibers which originate in the ganglion cell layer of the retina and converge upon the nerve head from all points in the fundus. At the surface of the nerve head, these axons bend acutely to leave the globe through a fenestrated sclera canal called the lamina cribrosa. Intra ocular portion of the optic nerve head has a diameter which varies from 1.18mm to 1.75 mm with average of 1.5mm.

DIVISIONS OF THE OPTIC NERVE HEAD

1. SURFACE NERVE FIBRE LAYER: The innermost portion of the optic nerve head, composed predominantly of neurons.

2. PRELAMINAR REGION: This layer begins at the posterior limit of the superficial nerve fibre layer and ends where the neurons pass through the lamina cribrosa. Predominant structures at this level are neurons and astrocytes.

3. LAMINA CRIBROSA REGION: This portion contains fenestrated sheets of scleral connective tissue and occasional elastic fibres. Fascicles of neurons leave the eye through these openings.

4. RETROLAMINAR REGION: This is the part of the optic nerve head posterior to the laminar region. This area is charecterised by a decrease in astrocytes and the acquisition of myelin that is supplied by the

oligodendrocytes. The posterior extent of the retro lamina region is not clearly defined.[1,3]

BLOOD SUPPLY OF THE OPTIC NERVE HEAD:

ARTERIAL SUPPLY

1. SURFACE NERVE FIBRE LAYER is mainly supplied by the arteriolar branches of the central retinal artery, which anastomose with vessels of Pre-laminar region and are continuous with the peripapillary retinal and long radial peripapillary capillaries.
2. PRELAMINAR and LAMINAR are supplied primarily by short posterior ciliary arteries which form a perineural circular arterial anatomosis at the sclera level called circle of Zinn –Haller. The branches from this circle penetrate the optic nerve to supply the pre-laminar and the laminar regions along with the peripapillary choroid.
3. RETRO-LAMINAR REGION is supplied by both the ciliary and retinal circulation with the former coming from recurrent pial vessels.

Peripheral centripetal vascular system formed by pial branches of the peripapillary choroid , arteries or Zinn , central retinal artery and

ophthalmic artery. Axial centrifugal vascular system formed by branches, from the intraneural part of the central retinal artery.

VENOUS DRAINAGE

The venous drainage of the optic nerve head is mainly by the central retinal vein. The pre-laminar region is also drained by the choroidal veins.[1,4]

AXONS IN OPTIC NERVE HEAD:

The arcuate fibers occupy the superior and inferior temporal portions of the optic nerve head, with axons from the peripheral retina taking a more peripheral location. The arcuate fibers are more susceptible to early glaucomatous damage. The papillomacular fibers spread over approximately one third of the distal optic nerve, primarily inferior temporally where the axonal density is higher. They intermingle with extramacular fibers, which may explain the retention of central vision during early glaucomatous optic atrophy.[1,4]

INTRAOCULAR PRESSURE

Normal intraocular pressure may be defined as that pressure that doesnot lead to glaucomatous damage of the optic nerve head. Unfortunately, such a definition cannot be expressed in precise numerical terms, in that all eyes do not respond the same to given pressure levels.

Three factors determine IOP :

- Rate of aqueous humor production
- Resistance to aqueous outflow across trabecular meshwork to schlemm's canal
- Level of episcleral venous pressure

FACTORS EXERTING LONG-TERM INFLUENCE ON IOP

1. GENETICS: The IOP within the general population appears to be under hereditary influence, through a polygenic, multifactorial mode of inheritance.

2. AGE: There is an increase in IOP with age.

3. GENDER: IOP is equal between the sexes in the agegroup of 20-40 years. In older age groups, the apparent increase in mean IOP with age is greater in women.

4. REFRACTIVE ERROR: A positive correlation exixts between IOP and both axial length of the globe and increasing degrees of myopia.

5. ETHNICITY: Blacks have higher IOP than whites.

FACTORS EXERTING SHORT-TERM INFLUENCE ON IOP

1. DIURNAL VARIATION: The intraocular pressure is subject to cyclic fluctuations throughout the day. The reported mean amplitude of daily fluctuation ranges from approximately 3mm Hg to 6 mm Hg. An amplitude greater than 10 mm Hg is generally considered pathologic. Many people reach their peak pressures in the morning hours, but others do so in the afternoon, in the evening or during sleep. The primary clinical value of measuring diurnal IOP variation is to avoid the risk of missing a pressure elevation with single readings. The intraocular pressures are recorded 6 times during the day at 4 hourly intervals and the graph is plotted connecting all points. No peak exceeding 21 mm of Hg confirms the diagnosis of normal tension glaucoma.[5]

2. POSTURAL VARIATION: The IOP increases from sitting to the supine position with reported average differences of 0.3-6 mm of Hg.

3. EXERTIONAL INFLUENCE: Prolonged exercise such as running lowers the IOP. Valsalva maneuver increases the IOP.

4. LID MOVEMENT: Blinking has been shown to raise the IOP.

5. INTRAOCULAR DISEASES: Anterior uveitis and retinal detachment are associated with a reduced IOP.

6. SYSTEMIC CONDITIONS: Systemic hypertension and hyperthermia are associated with elevated IOP. The IOP has been reported to be lower with hyperthyroidism and higher with hypothyroidism. Diabetic patients have been reported to have higher IOP than the general population.

7. ENVIRONMENTAL CONDITIONS: Cold air reduces the IOP.

8. GENERAL ANAESTHESIA: It is usually associated with reduction in IOP. Drugs like ketamine and succinyl choline cause a transient raise in IOP.

9. OTHERS: Alcohol and Heroin decrease the intraocular pressure whereas LSD and corticosteroids increase the intraocular pressure.[1]

PATHOGENESIS OF GLAUCOMATOUS OPTIC ATROPHY

The pathogenesis of glaucomatous optic atrophy has remained the matter of controversy since mid 19th century.

The mechanical theory was proposed by Muller in which the elevated IOP led to direct compression and death of the neurons.

The vasogenic theory was proposed by Von-Jaeger. According to this theory the structural and functional defects occurring in glaucoma are due to ischemia. The most elaborate support for this theory was advanced by HAYREH. He proposed that both an increase of IOP and fall of blood pressure lead to fall of perfusion in the ocular vessels. The fall of perfusion pressure can obliterate vessels first in the post laminar and retrolaminar region. The blood flow in the pre-laminar and post-laminar region and the choroid lack the ability of autoregulation. Optic cupping results from chronic ischemia of the optic nerve head.

There are two weak points in the above theory. According to HAYREH the primary site of axon damage is the prelaminar disc area, but it has been found that it is actually in the lamina cribrosa region. There is evidence in favour of effective autoregulation of blood flow in the optic nerve head. Thus, Glaucomatous damage to the optic nerve is multifactorial and is affected by more than just IOP elevation.[1]

CHARACTERISTICS OF GLAUCOMATOUS OPTIC ATROPHY

1. FOCAL ATROPHY

Selective loss of neural rim occurs primarily in the inferotemporal region of the optic nerve head. The temporal rim is typically involved after the vertical poles, with the nasal quadrant being the last to be involved. The focal atrophy of the neural rim often begins as a small discrete defect usually in the inferotemporal quadrant which has been called as polar notching.

2. CONCENTRIC ATROPHY

Glaucomatous damage less commonly results in concentric enlargement of the cup more often directed inferotemporally or superotemporally. Loss of neural rim usually begins temporally and then progresses circumferentially towards the poles . this is called as temporal unfolding.

3. DEEPENING OF THE CUP

Exposure of the underlying lamina cribrosa by the deepening of the cup is recognized by the gray fenestra of the lamina which is called as the laminar dot sign.

4. ADVANCED GLAUCOMATOUS CUPPING

If the progressive changes of glaucomatous optic atrophy are not arrested by appropriate measures to reduce IOP , the ultimate result is total cupping which is seen clinically as a white disc with loss of neural rim tissue and bending of all vessels at the margin of the disc. This has also been called as bean pot cupping, because the cross section of a histological specimen reveals extreme posterior displacement of the lamina cribrosa and undermining of the disc margin.[1,2,5,21]

VASCULAR SIGNS OF GLAUCOMATOS OPTIC ATROPHY

a. SPLINTER HEMORRHAGES : These are present near the margin of the optic nerve head and are a common feature of glaucomatous damage. They occur more commonly in patients with normal tension glaucoma. They tend to come and go so that they may be seen in one visit and be gone in the next only to reappear at a later date in the same or a new location. The most common location is the inferior quadrant, most often seen in the early to middle stages of glaucomatous damage. A thin neuroretinal rim was found to be a risk factor for the development of optic disc hemorrhages. They commonly occur with minimal pressure elevation or in eyes with normal-tension glaucoma.[24] They also occur more commonly on

diabetic patients with glaucoma. It should be viewed as a sign that the glaucoma may be out of control.[1]

b. BARING OF THE CIRCUMLINEAR VESSEL

In many normal optic nerve heads , one or two vessels may curve to outline a portion of the physiological cup. With glaucomatous enlargement of the cup, these circumlinear vessels may be bared from the margin of the cup.

c. NASALISATION OF VESSELS

Nasal displacement of the retinal vessels on the optic nerve head is a sign of glaucomatous cupping.

d. BAYONETING OF THE VESSELS

If a retinal vessel crosses the sharpened rim , it will bend at the edge of the disc creating the bayoneting sign.[1,2]

VISUAL FIELD LOSS IN GLAUCOMA

1. PERIPHERAL LOSS

Defects along the peripheral boundaries of the visual field eg; peripheral nasal steps, vertical steps and temporal sectoral defects are most often found in association with early glaucomatous visual field loss.

2. LOCALIZED NERVE FIBER LAYER DEFECTS:

The glaucomatous process causes initial damage to one or more axon bundles creating a localized visual field defect.

3. ARCUATE DEFECTS

An arcuate visual defect is strongly suggestive of glaucoma. This arcuate scotoma starts from the blindspot and arches above and below fixation or both to the horizontal median raphe, corresponding to the arcuate retinal nerve fibers.

As the field defects develop within the arcuate area they most often appear as paracentral scotomas. Occasionally the early arcuate defect may connect with the blind spot and taper to a point in a slightly curved course which has been called as Seidel scotoma. As the isolated defects enlarge and coalesce, they form an arching scotoma that eventually fills the entire arcuate area from the blind spot to the median raphe , which is called as

Bjerrum scotoma. With further progression , a double arcuate or ring scotoma develops.

4. NASAL STEP

Unequal contraction on the peripheral side of the defect due to loss of corresponding bundles of peripheral fibers produces the peripheral nasal step of Ronne.[1,2,5,11]

5. EARLY GLAUCOMATOUS FIELD DEFECTS

a. CONCENTRIC CONTRACTION

Isopter contraction is more often marked in the nasal field called as crowding of nasal isopters is a early field defect in glaucoma.

b. ANGIOSCOTOMATA

These are long branching scotomas above and below the blindspot which are presumed to result from shadows created by the large retinal vessels .[1]

6. ADVANCED GLAUCOMATOUS FIELD DEFECTS

The natural history of progressive glaucomatous field loss is eventual development of a complete double arcuate scotoma, which coalesce nasally at the horizontal raphe and may extend to the peripheral limits in all areas except the temporal side . This results in a central island and a temporal island in advanced glaucoma.[1,2]

CLASSIFICATION OF GLAUCOMAS

The major classification of the glaucomas relates to the configuration of the anterior chamber angle and the age of onset of the disease. Glaucomas are classified into:

1. Open angle glaucoma-
    a. Primary open angle glaucoma
    b. Normal tension glaucoma
    c. Juvenile glaucoma
    d. Glaucoma suspect
    e. Secondary open angle glaucoma
2. Angle closure glaucoma-
    a. Primary angle closure glaucoma
    b. Secondary angle closure glaucoma either with or without pupillary block
3. Childhood glaucoma-
    a. Primary congenital or infantile glaucoma
    b. Glaucoma associated with congenital anomalies
    c. Secondary glaucoma in infants and children.[1]

NORMAL TENSION GLAUCOMA

Normal tension glaucoma is defined as a condition in which cupping of the optic nerve head , loss of the neuroretinal rim and visual field defects similar to other forms of chronic glaucoma in which IOP level outside the statistically normal range without treatment has not been documented nor is any other cause for these changes apparent.[1]

Normal tension glaucoma(NTG) also called low tension glaucoma, is a diagnostic and therapeutic conundrum. NTG is classified into progressive and non-progressive forms.[1,28]

Non- progressive NTG is usually due to transient hemodynamic catastrophe and is actually a form of pseudo NTG. Progressive NTG may worsen more rapidly than primary open- angle glaucoma despite medical and surgical treatment.[1]

Both the eyes are affected in approximately 70% of the patients, and the disease is more commonly seen in patients over the age of 60.[39,40] Women are affected about twice as often as men and NTG represents 10-30 % of all forms of glaucoma.[1,38]

Open angle glaucoma is a spectrum of disorders in which elevated IOP is the most influential and glaucomatous optic atrophy predominate at the other end. Controversy remains about whether NTG represents a

distinct disease entity or is simply POAG with IOP within the average range.

In normal tension glaucoma risk factors other than IOP may play an important role. Many authorities have hypothesized that local vascular factors may have a significant role in the development of this disorder.

Normal tension glaucoma is divided into two groups:

1. Senile sclerotic group:

   This constitutes old patients with vascular disease and is characterized by shallow sloping of neuroretinal rim.

2. Focal ischemic group:

This constitutes relatively younger patients with deep, focal polar notching of the neuroretinal rim.[1]

DIFFERENCE BETWEEN NTG AND COAG

Clinical differences between NTG and COAG are as follows,

1. The neural rim has been found to be significantly thinner in NTG than in COAG, especially inferiorly and inferotemporally.[19]
2. Cupping in NTG is found to be more broadly sloping than in COAG.
3. Optic disc hemorrhages are more prevalent in NTG.
4. Localized retinal nerve fiber layer defects are found in NTG as compared to diffuse nerve fiber layer defects in COAG.
5. Patients with NTG have larger peripapillary atrophy particularly in zone beta than in COAG.
6. NTG patients have deeper and more localized scotomas .[23]
7. Higher incidence of proximity of scotomas to fixation in NTG.[1]

Although NTG is distinguished from COAG by an IOP that is never recorded to exceed 21mm Hg , the pressures do tend to be higher than those in the normal population. An IOP reduction of atleast 30% is significantly associated with protection of visual field and nerve status . Hayreh suggested that NTG differs from anterior ischemic optic neuropathy only in that the latter is a more acute process.[1]

ASSOCIATED RISK FACTORS

NTG can be mimicked by many conditions. Elevated IOP can be obscured in patients with systemic medical beta blockers. NTG patients have significantly greater nocturnal blood pressure drops than normal patients. In normal tension glaucoma, there are two aspects to the relationship between systemic blood pressure and optic nerve damage. One is whether patients with normal tension glaucoma have chronic blood pressure abnormalities. The other is the possible role of episodes of acute hypotension in patients with normal tension glaucoma.[1,21]

A greater lack of autoregulation of optic nerve head circulation is noted in NTG. Patients with NTG have an increased frequency of headaches with or without migraine. An abnormally reduced blood flow in the fingers in response to exposure to cold have been found.[1]

Hematologic abnormalities like increased blood and plasma viscosity and hypercoagulability. Hypercholesterolemia has also been reported to be higher among patients with NTG. An increased incidence of paraproteinemia and autoantibodies including antirhodopsin antibodies and anti- glutathione S tranferase are present in NTG.[1,21]

CLINICAL PRESENTATION

Patients may complain of reduced vision or other visual difficulties resulting from extensive visual field loss. More often the abnormal appearance of the optic nerve head is noted during examination of an asymptomatic patient. Because these patients have normal IOP and good central vision the disease is often missed when proper evaluation of the optic nerve head is not done. Routine use of slit lamp with Hruby lens or handheld 78 D or 90 D lens increases the likelihood of detecting cupping.

EVALUATION OF NORMAL TENSION GLAUCOMA

Before making a diagnosis of NTG, clinician should measure the patient's intraocular pressure at various times during the day. Since the mathematical calculation for Goldmann Applanation tonometer is based on a presumed average central corneal thickness(CCT) , variations in this parameter can lead to errors in the measurement. Variations of CCT in normal corneas can lead to falsely high IOP with thicker corneas and falsely low IOP recordings in thinner corneas.[1]

Gonioscopy to be performed to rule out angle closure, angle recession and previous signs of inflammation. Careful stereoscopic disc evaluation is essential to rule out other congenital or acquired disc anomalies, such as optic nerve coloboma, drusen or enlarged cup.

Clinician must also consider the patient's medical history, any record of cardiovascular disease and low blood pressure caused by hemorrhage, myocardial infarction or shock. If diagnosis couldnot be established or if findings are atypical, neurological evaluation to be done.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of normal tension glaucoma includes,

1. Congenital disorders: Optic nerve pit or coloboma, Autosomal dominant optic atrophy.[18,22]

2. Acquired disorders :

   - Past history of steroid use by any route which may have led to elevated IOP
   - Past history of trauma or surgery that may have led to elevated IOP
   - Hemodynamic crisis
   - Methyl alcohol poisoning
   - Optic neuritis
   - Anterior Ischemic optic neuropathy
   - Non arteritic ischemic optic neuropathy
   - Compressive lesions of the optic nerve and tract
   - Trauma
   - Wide diurnal fluctuation in IOP[1,33]

MANAGEMENT

Although damage to the optic nerve head and visual field may progress even at low normal pressures in normal tension glaucoma, there is compelling evidence for further IOP reduction in this disorder. An additional aspect in the management of Normotensive glaucoma is the treatment of cardiovascular abnormalities like anemia, congestive heart failure, transient ischemic attacks and cardiac arrhythmias to ensure maximum perfusion of the optic nerve head.

Therapy for NTG can be difficult and controversial. The goal of therapy should be to achieve an IOP as low as possible without inducing complications.

Systemic medications such as calcium channel blockers are advocated because of the possible beneficial effects of increasing capillary perfusion of the optic nerve head. If systemic treatment with calcium channel blockers is undertaken, it should be co-ordinated with the patient's primary care physician because of the possible side effects.

Medical therapy is the most common initial approach in treating NTG. As with all glaucomas, it is useful for the ophthalmologist to change or add medications to one eye at a time so that contralateral eye can be used as a control to assess therapeutic response. More than 25% of baseline pressure needs to be reduced to assess the progression.

The Beta blockers were also tried in normal tension glaucoma. Timolol was found to have no change in reduction of ocular perfusion at night. Levobunol and Betaxolol decrease the intraocular tension and also have a unique property of increasing the pulsatile ocular blood flow to the optic nerve.

Latanoprost causes IOP reduction through out the night by increasing the uveoscleral out flow . 20-30% IOP reduction is attained and it causes7.9% increase in perfusion pressure of the optic nerve head. Bimatoprost increases the optic nerve head perfusion.Travoprost is similar and comparable to latanoprost.

Brimonidine is an Alpha –2 adrenergic receptor agonist and has been proposed to help in NTG by its newer mode of action (decreased aqueous production & increased trabecular outflow). Secondly it a neuroprotective it protects the retinal ganglion cells.

Calcium channel Blockers block membrane bound calcium channels & inhibit calcium influx causing relaxation of smooth muscle cells in vascular walls , a decrease in vascular tone & improvement in blood flow. Flunarizine improves blood flow in NTG. Nimodepine & Nilvadepine show beneficial effects in NTG patients. It is still under research process.

Other Neuro supportive drugs such as Methycobalamine has been widely used now and proved to be effective. Their exact mechanism of action is not known.

If medications are ineffective in controlling the disease, laser trabeculoplasty can be tried. Glaucoma filtering surgery may be indicated in an attempt to obtain the lowest IOP. It carries risk of postoperative hypotony. Hence the option is preserved for those patients with undoubted field progression .Surgery should not be performed on making the diagnosis.

When surgical management is indicated, trabeculectomy with mitomycin c is preferred. Antifibrotic agents may be used to improve the success rate of filtering surgery and to reduce the postoperative and long term IOP in these patients with low target IOPs. The goal of treatment in normotensive glaucoma is to achieve a targeted pressure reduction of around 30% from the initially detected intraocular pressure.[1]

Collaborative Normal Tension Glaucoma Trial (CNTGS):

The aim of the study was to determine if IOP lowering treatment is effective in reducing the progression of NTG. 140 patients randomized to receive either medication /surgery (End point was reduction of IOP by 30%) or to have no treatment. The untreated eyes had 35% chance of progression compared to 12% chance of progression in the treated group. Collaborative normal tension glaucoma study concluded that IOP reduction in normal tension glaucoma decreases glaucoma progression.[1]

ASSESSMENT OF FIELDS BY AUTOMATED PERIMETRY

Automated perimetry is accepted as the standard way of measuring the visual field. The standard protocol of static white on white stimuli is called as standard automated perimetry. A major limitation of tangent screen and arc erimeters was a lack of standardization of the test objects and the background as well as the patient's fixation.

The computers have provided the new capability that was not possible with the manual perimetry, including random presentation of targets, estimation of patient's reliability, reduced variability and statistical evaluation of data at many levels. The introduction of threshold strategies makes it more accurate, reliable and faster than manual perimetry.

Static targets are most commonly used in automated perimetry. The targets may either be projected into the bowl which is the current standard, or illuminated from light emitting diodes or fiberoptics. The standard stimulus in most automated perimeters is a white light on a white background which tests the patient's differential light sense.

With each Octopus model the stimuli are projected onto the bowl and fixation is monitored by both the corneal light reflex method and a television view of the patient's eye.

TEST PATTERNS

The central 24-30 degrees field with 6 degree separation between test locations is the commonly used test pattern. The 6 degree grid may miss the physiologic blind spot as well as some glaucomatous field defects and hence tighter grids are suggested for meticulous testing of fields. Static testing of the peripheral nasal fields provides a valuable information in detecting glaucomatous defects.

TESTING STRATEGIES

All fully automated perimetries take the advantage of the computers by using random presentation of the targets to avoid patient anticipation of the next presentation sites. An additional technique called as adaptive technique is used, in which stimuli are presented according to the presumed normal threshold contour based on age corrected normal data and patient's response to preliminary spot tests. Fully automated perimeters provide suprathreshold and full threshold measurements.

FULL THRESHOLD PERIMETRY

Threshold static perimeters are capable of a variety of testing strategies. The most commonly used programs measure the retinal threshold at 70-80 points within the central 24-30 degrees . a suprathreshold target is first presented and then it's luminosity is

gradually increased or decreased until the patient's threshold is crossed . the threshold is then crossed a second time with smaller increments of change in luminosity to refine the threshold determination.

TENDENCY ORIENTED PERIMETRY

It is another fast strategy algorithm available on new Octopus perimeters. It also uses a computational approach to estimate threshold values by extrapolating information from surrounding test points. The mean testing time for the TOP strategy was slightly more than 2 ½ minutes, compared to approximately 4 minutes for SITA fast.

FASTPAC

Another threshold testing strategy to reduce testing time is the FASTPAC program of the Humphrey field analyzer , which estimates thresholding from a single threshold crossing in 3db increments, in contrast to standard double threshold crossing with 4db and 2 db.

SWEDISH INTERACTIVE THRESHOLD ALGORITHM

This algorithm significantly minimizes test time without significant reduction of data quality. Two versions of SITA are currently available: SITA standard and SITA fast.

INTERPRETATION OF RESULTS

With most full threshold programs , a percentage of random locations are retested to determine the reproducibility at those points. The patient's general reliability is assessed with a series of false-positives (patient responds when no target is presented) and false negatives (patient fails to respond to a stimulus of maximum intensity where a stimulus was previously reported to be seen), as well as the frequency of fixation losses and the number of stimuli required to complete the test.

The Octopus also provides printouts with probability, corrected probability, comparison and corrected comparison.

Global indices are the mathematically analyzed data allowing detection of more subtle visual field abnormalities. The average of all points in the comparison plot gives the mean defect. These indices primarily reflect diffuse changes. The way to detect localized defects is to calculate the number of threshold values that deviate significantly from the age corrected normal which is called as loss variance. Corrected loss variance takes into account the short term fluctuations.[1]

ROLE OF OPTICAL COHERENCE TOMOGRAPHY IN GLAUCOMA

Optical Coherence Tomography (OCT) was pioneered by Fujimoto and others at Massachesetts eye institute. OCT was first applied in the eye by Puliafito in 1986. It was developed as a commercial product by Humphrey division of Carl Zeiss.

OCT is a non invasive cross-sectional diagnostic imaging modality that is capable of producing highly accurate structural representations of the retinaand the posterior segment tissues. The resolution limit of OCT is 8 micron. A highly reflective red layer at the vitreoretinal interface corresponds to the retinal nerve fiber layer.[1,9]

The principle of OCT involves a low- coherence infrared diode laser source, which is divided into reference and sample paths. Reflected sample light from the subject's eye creates an interference signal with the reference beam, which is detected in a fiberoptic interferometer. Cross-sectional images of the retina and disc are then constructed from a sequence of signals.

The straight blue line which connects the edges of the RPE represents the disc diameter. Automated determination of the disc margin as the end of the RPE was used for this analysis. The cup diameter is

denoted by a parallel red line constructed 150 micron anterior to disc diameter. The structures below the red line are defined as the cup. Structures above the red line, the neuroretinal rim. Three dimensional spectral domain (Fourier domain OCT) gives faster and more accurate results.[17]

Optic nerve head analysis is performed with a fast optic nerve scan protocol. Six radially linear scans centered on the ONH were analysed cross-sectionally. The Retinal nerve fiber layer thickness(RNFL) measured be averaging the results of 3 sequential circular scans with diameter of 3.4 mm centered at ONH using fast RNFL scan mode. RNFL thickness is determined by the difference in distance between the vitreoretinal interface and a posterior boundary based on a predefined reflectivity signal level. RNFL thickness is determined at 256points covering a set diameter (3.4 mm) around the center of the optic disc 3 times.

The high axial resolution of OCT allows for direct measurement of RNFL thickness with micron scale accuracy. Radial scans through the disc provide contour information that is valuable in objectively assessing optic disc cupping and NRR thinning.

In OCT, A scan was defined by a sequence of data points typically acquired in order of increasing depth. B and C scan images are defined as a matrix of pixels, typically acquired either by rows or columns.

The line graph displays RNFL thickness on the vertical axis and A scan location on the horizontal axis. The display of the RNFL thickness along the scanned circle begins temporally at 0 degree. The RNFL thickness profile is then plotted in a clockwise direction for the right eye and counterclockwise direction for the left eye. In line graph presentation, the percentile value derived from the normative database are represented as colour bands.

The pitfalls of this imaging are , the image quality is dependant on the operator skill, patient related factors like the pupil diameter and media clarity.

However NFL thinning can often be detected before visual field loss or progression of cupping is identified. Clinically, cases of advanced glaucoma exhibit direct correlation between areas of NFL thinning and visual field defects. Objective assessment of glaucomatous change by OCT may clarify the subjective variability of visual field testing and biomicroscopic nerve head evaluation. [1,9]

FUNDUS FLUORESCEIN ANGIOGRAPHIC STUDIES IN GLAUCOMA

Normal flourescein pattern of the optic nerve head is usually described in three phases.

1. An initial filling or preretinal arterial phase- represents the filling of prelaminar and laminar regions by posterior ciliary arteries.
2. The peak fluorescence or retinal arteriovenous phase – represents the filling of the dense capillary plexus on the nerve head.
3. A late phase consists of delayed staining of the optic nerve head – represents the flourescein in the connective tissue of the lamina cribrosa.

The glaucomatous eyes reveal two types of filling defects.

- Persisting hypoperfusion or absolute filling defects
- Transient hypoperfusion or relative filling defects

Persisting hypoperfusion or absolute filling defects are more common in normal tension glaucoma. They are said to correlate with visual field defects. The filling defect may either be focal or diffuse. Focal defects occur in inferior or superior poles of optic nerve head. In glaucomatous eyes they are most seen in the wall of the cup. Focal defects are common in normal tension glaucoma.[32]

In addition, in normal tension glaucoma, the choroidal phases are delayed and retinal arterio-venous phase times are prolonged possibly from the increased resistance in the central retinal and posterior ciliary arteries.[1,25,30]

TRANSCRANIAL DOPPLER ULTRASOUND OF OPHTHALMIC ARTERY

Transcranial Doppler ultrasound is used to study non-invasively the blood flow velocity of intracranial vessels including the ophthalmic artery flow velocity. It consists of a 2 MHz pulsed Doppler with a fast Fourier transformation which is used to derive and analyse the spectrum of returning echoes of various frequencies. The peak systolic velocity, mean envelope velocity, end diastolic velocity and the resistivity index are measured.[28]

The latest is the confocal scanning laser Doppler flowmetry. Normal tension glaucoma is characterized by reduced blood flow in the peripapillary retina, suggesting that blood flow deficits accompany and may contribute to the disease development in these patients.[29]

AIMS OF THE STUDY

1. To analyse the clinical presentation, risk factors and intraocular pressure level in normal tension glaucoma.
2. To detect the field defects by automated perimetry and to correlate them with the retinal nerve fiber layer thickness measured by OCT.
3. To evaluate the response to treatment and follow up of the patients to detect progression or regression in the field defects.
4. To find the fundus flourescein angiographic studies of the optic nerve head and retina in normal tension glaucoma.

INCLUSION CRITERIA

1. Patients without lens changes or minimal lens changes were taken.
2. Patients with gonioscopically open angles
3. Intraocular pressure < 21 mm of Hg (after Central corneal thickness correction).

EXCLUSION CRITERIA

1. Patients with dense lens changes.
2. Intraocular pressure > 21 mm of Hg even on one instance during diurnal variation test.
3. Presence of peripheral anterior synechia in gonioscopy.

MATERIALS AND METHODS

The study was a prospective case series conducted at Glaucoma services at Regional Institute of Ophthalmology from the period between July 2007 – July 2009. The study was done in 100 patients (198 eyes) of established Normal tension glaucoma after complete evaluation

CLINICAL EXAMINATION

The clinical assessment of the patient began with a thorough history taking about defective vision, defective field of vision, frequent change of glasses were enquired. Any serious operation or accident with significant blood loss, cardiac arrest, myocardial infarction, migraine, diabetes, hypertension, hypotension, poor nutrition, Raynaud's phenomenon, other connective tissue and autoimmune disorders, tobacco or alcohol use and neurological problem were enquired about. Family history of glaucoma , any prior head trauma or eye injury , prior cataract surgery, history of anti-glaucoma drug usage, history of steroid usage either systemically or topically were also enquired.

Visual acuity was recorded and refraction was done in all cases to correct refractive errors. Ocular examination of both eyes with slit lamp bio-microscope to detect any features of secondary glaucoma and lens

changes were done. Gonioscopy was done and the angles were graded by using Shaffer's grading method.

Intra-ocular pressure was measured using Goldmann Applanation tonometer and the Central corneal thickness was measured by Pachymetry. The values of Goldmann tonometer readings were taken as such for central corneal thickness (CCT) values between 510 -530 microns. For readings beyond that, correction factor of 3mm for every 50 microns[1], so that IOP + 3 mm of Hg for every 50 microns decrease in CCT(in thinner corneas) and – 3mm of Hg for every 50 microns increase in CCT (in thicker corneas) was taken.

Diurnal variation test (phasing) was done in all patients by recording 6 readings each 4 hours apart throughout the day and plotting the graph connecting all the 6 points such that none of the recordings were beyond 21 mm of Hg. The recording was done using Perkins applanation tonometer.

Fundus examination was done using + 90 D slit lamp biomicroscopy and the cup: disc ratio was noted. Associated findings like Nasalisation,Bayonetting, Laminar dot sign, Baring of circumciliary vessels and Splinter hemorrhages near the disc were noted.

Field charting was done by computer assisted static Automated perimetry (Octopus 123 , G1 x program, TOP strategy) for both the eyes. Reliable field testing with false positives and false negatives below 30 percent were taken for the study.

Optical Coherence Tomography was done in all patients to record the retinal nerve fiber layer thickness in all the four quadrants.

All the patients were started on 0.15% Brimonidine eye drops (alpha 2 agonist) twice a day and oral neurovitamins once daily.

The patients were followed up based on the fundoscopy and the extent of their field defects after 3 months and 6 months. Intraocular pressure recording, fundoscopy, field testing by automated perimetry was done in every visit. The response of intraocular pressure to medical treatment was noted. Fundus changes were recorded.

Any progression or regression or static nature of the field defect were noted. The patients were asked to either continue Brimonidine eye drops or substituted with Betaxolol or Latanoprost based on their response to therapy. The patients were reviewed after 3 months for repeat IOP and field testing. A decision whether to continue medical treatment or surgical treatment was then taken.

Blood pressure measurement, Hemoglobin and Lipid profile was done in all patients. Thorough cardiology and neurology evaluation was done for all the patients.

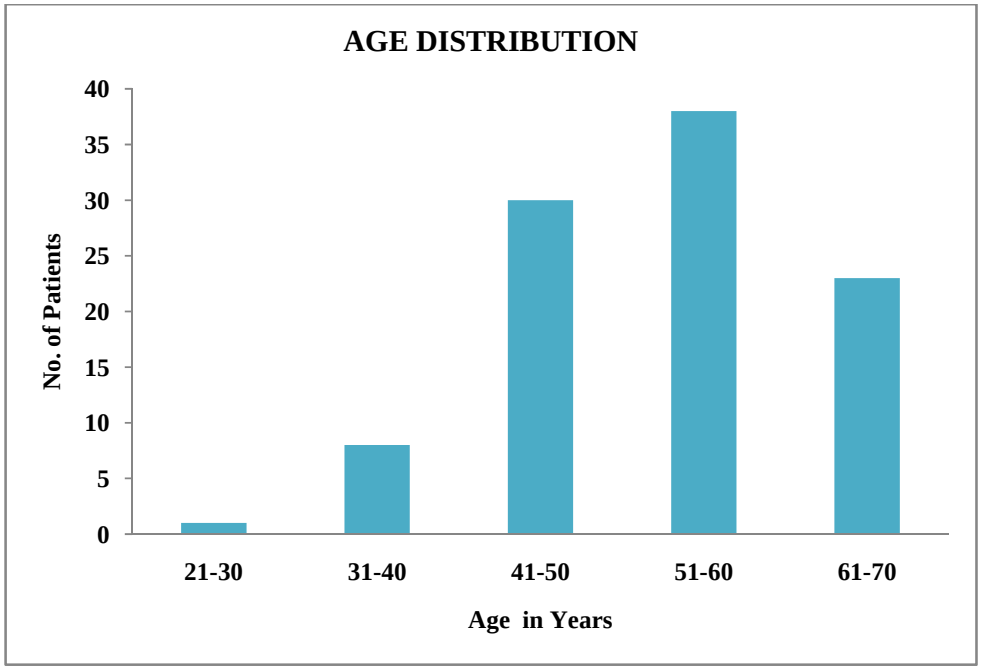
15 cases of Normal tension Glaucoma were taken after informed consent for Fundus Fluorescein Angiography of the retina and the optic nerve head. Patients with renal failure or cardiac failure were excluded from the test. The procedure was explained to the patient.The choroidal filling time, arterio-venous transit time and the filling defects on the optic nerve head were noted.

OBSERVATION AND ANALYSIS

198 eyes of 100 patients with intraocular pressure < 21 mm of Hg were taken for the study. 2 patients were one eyed, 1 had leucomatous corneal opacity and the other patient had evisceration done in one eye.

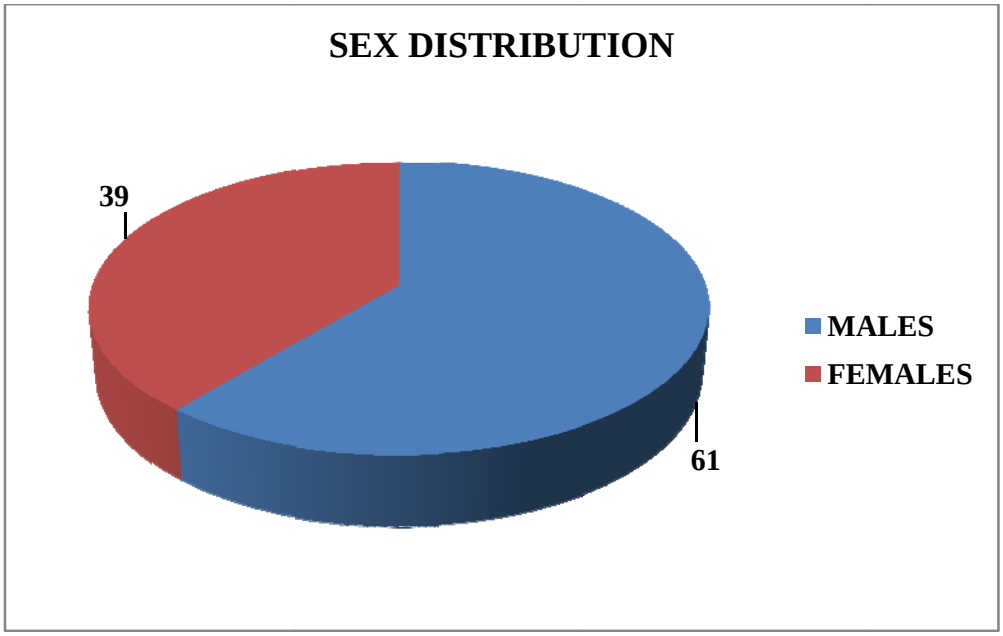
1. AGE DISTRIBUTION

The ages of patients studied varied from 21- 70 years. The average age incidence was 41-60 years. This is because most of this age group patients come for ophthalmic examination either for refractive error or defective vision due to lens changes. Thereby, normal tension glaucoma is detected by fundus examination and intraocular pressure measurement.



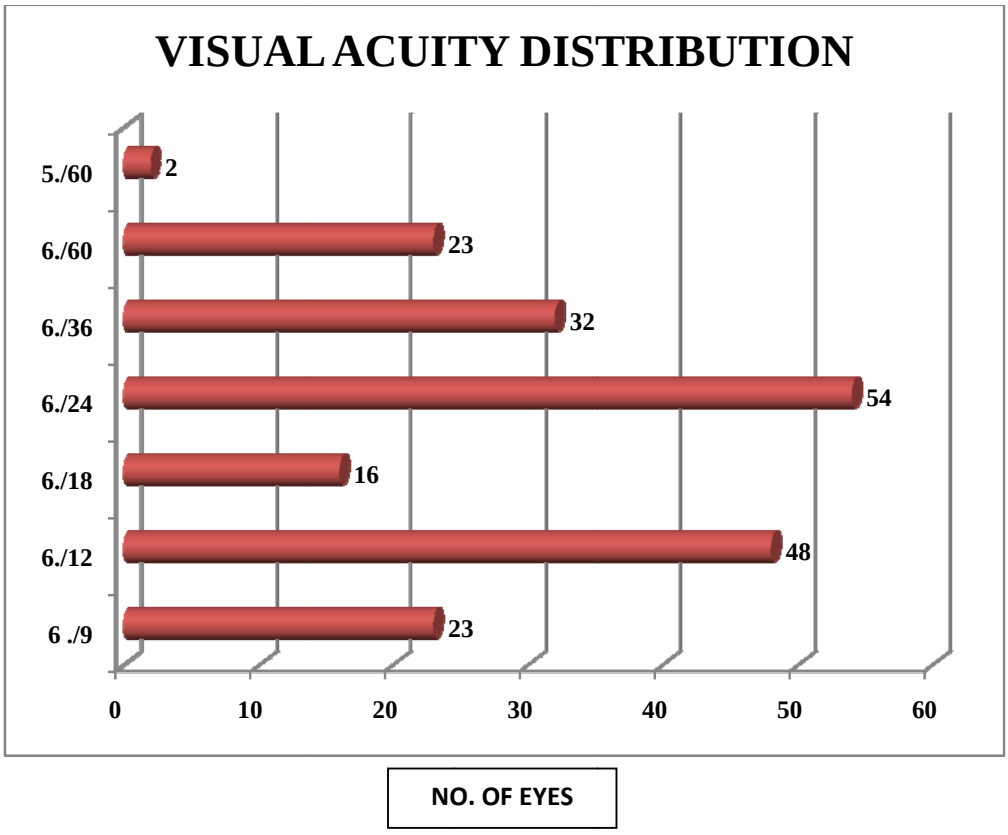
2. SEX DISTRIBUTION

In the study group, 61% were males and 39% were females. This is due to the awareness among male population regarding eye diseases and the need for spectacle correction for their work purpose. Hence detection of glaucoma is more in men in our country. Previous studies show that the incidence is more in females.[1,40]



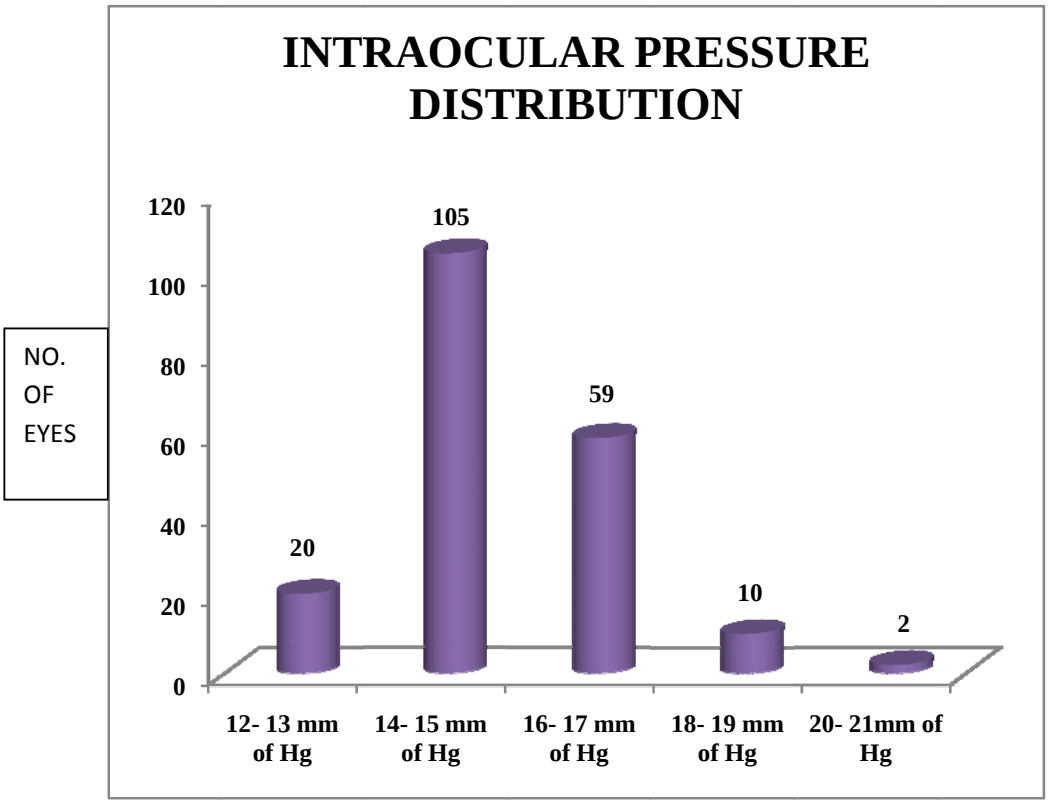
3. VISUAL ACUITY DISTRIBUTION

171 eyes (86%) presented with visual acuity of 6/36 and above. 23eyes(11%) had visual acuity of 6/60 and 2 eyes(1%) presented with 5/60 . The dimunition in visual acuity was due to refractive error in 132 eyes(66%) and due to early lens changes in 53 eyes(26%) and due to posterior capsular opacification in 12 eyes(6%). The defective vision was the reason that these patients presented to the hospital. Hence the presence of normal tension glaucoma was detected incidentally in these patients.

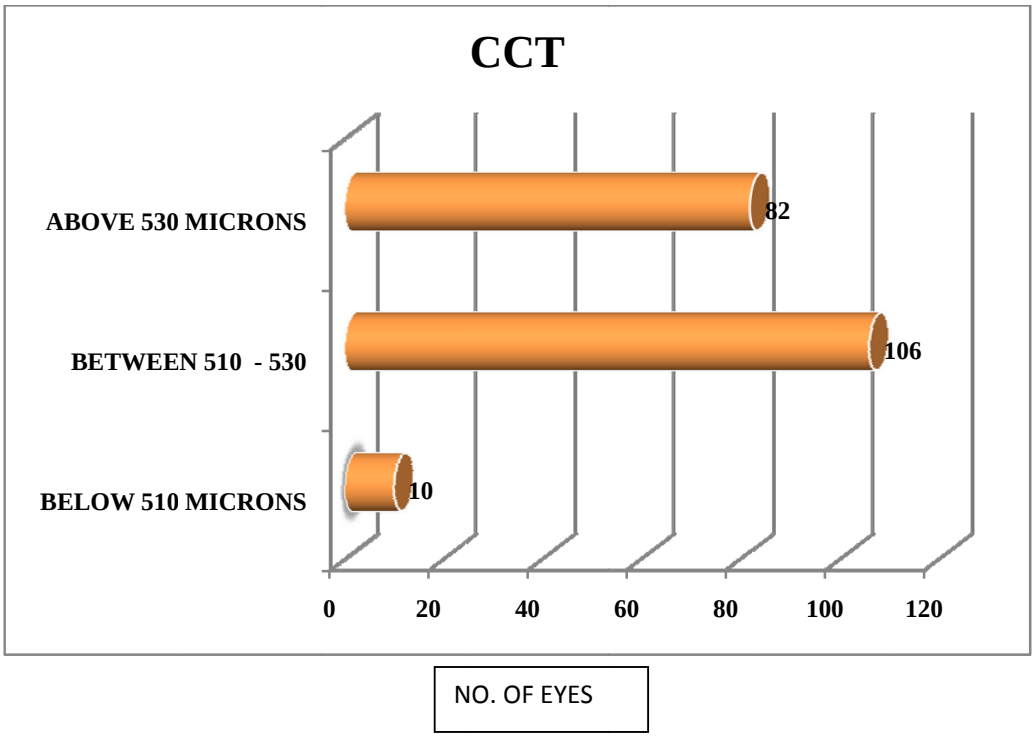


4. INTRAOCULAR PRESSURE DISTRIBUTION

The intraocular pressure was in the low teens and mid teens(12-15 mm of Hg) in 125 eyes(63%) and was in the high teens (16-19 mm of Hg) in 69 eyes (34%). 2 eyes had 20-21 mm of Hg on presentation. The mean intraocular pressure in this series is 15 mm of Hg. On the other hand, some studies indicate that patients with normal tension glaucoma tend to have intraocular pressures at the high range of normal.[1,41]

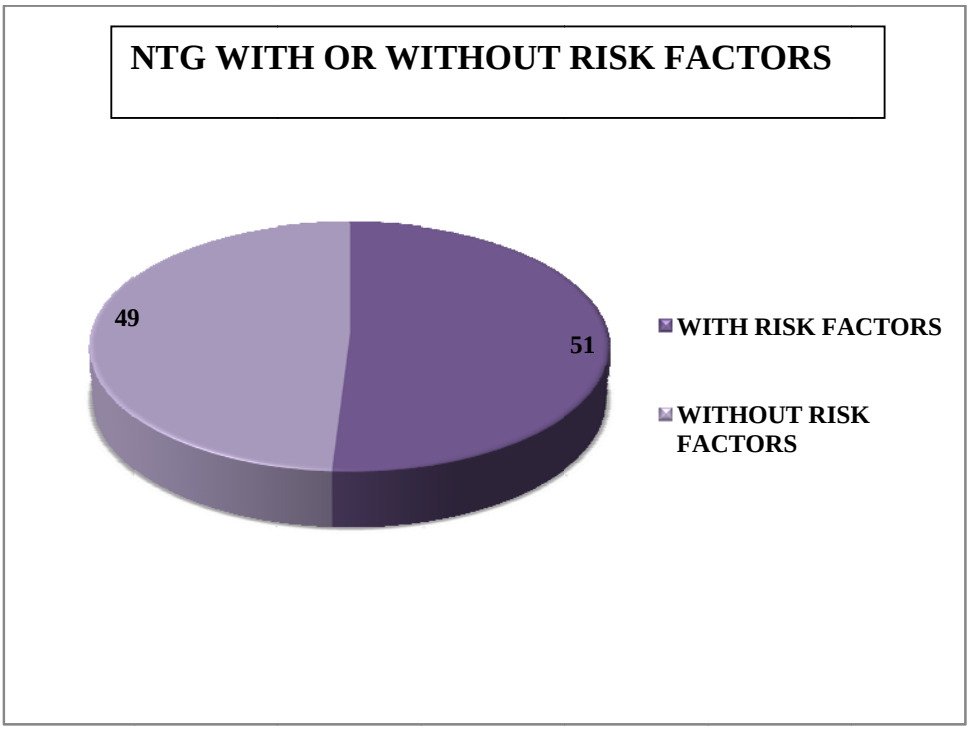


5. CENTRAL CORNEAL THICKNESS

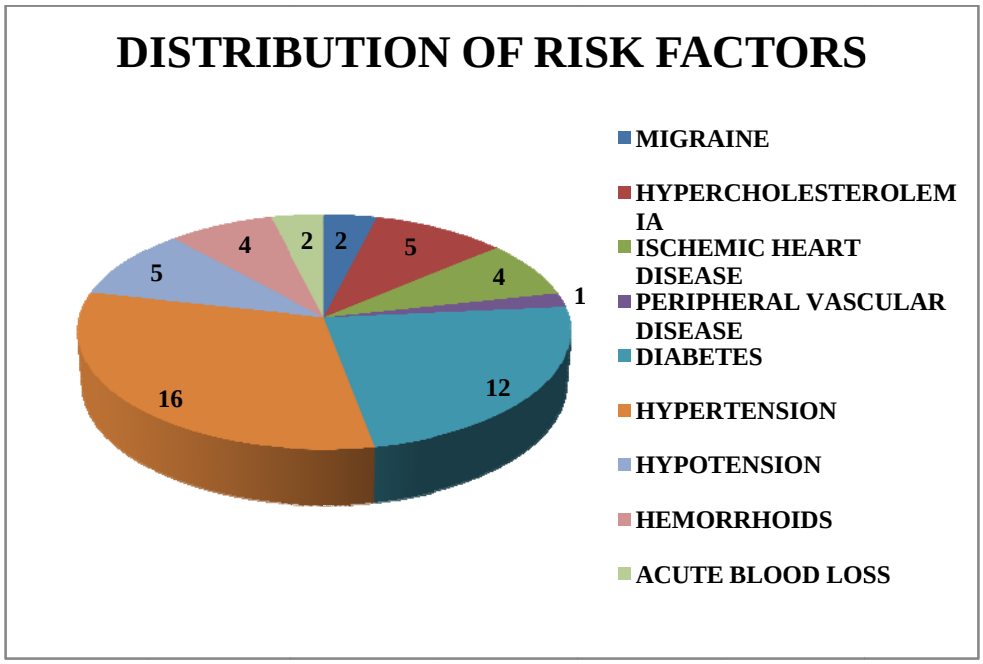


In our study, 106 eyes(53%) had central corneal thickness within the normal range(between 510-530 microns) and 82 eyes(41%) had CCT above 530 microns and 10 eyes(5%) had CCT below 510 microns. This is not in accordance with other studies which report CCT values lesser than normal(thinner corneas) in Normal tension glaucoma patients.[1]

6. ASSOCIATED RISK FACTORS



Of the 100patients taken for the study, 51 patients had associated risk factors. 16 patients had hypertension for which they were on systemic beta blockers, 5 had hypotension and history of acute blood loss in 4 patients with hemorrhoids and 2 patients had met with a road traffic accident. In normal tension glaucoma there are two aspects to the relationship between systemic blood pressure and optic nerve damage- chronic blood pressure abnormalities and role of chronic or acute hypotension due to an episode of acute blood loss.[1]



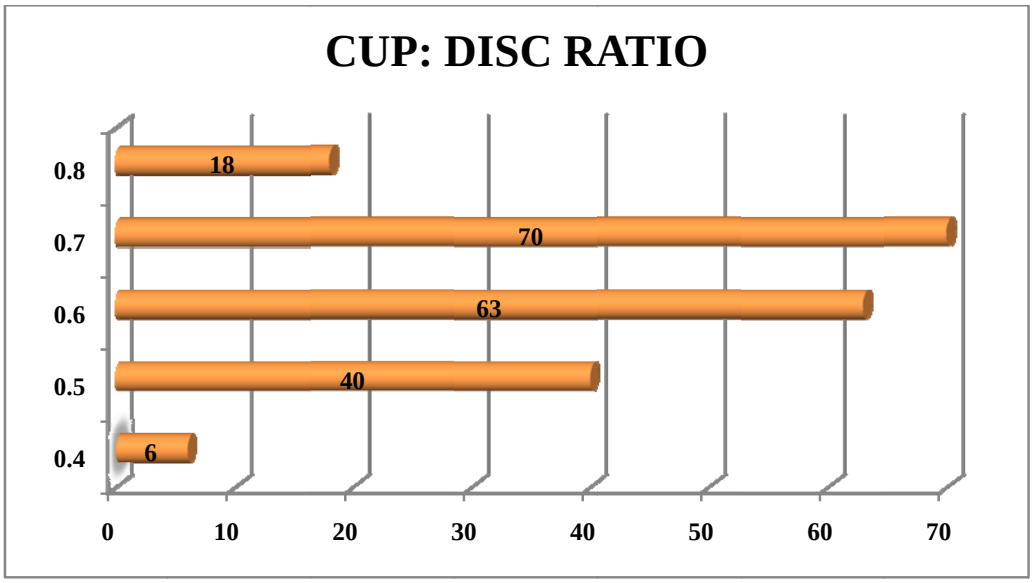
12 patients had diabetes , 5 had hypercholesterolemia with elevated lipid profile(increased serum cholesterol), 4 had ischemic heart disease and 1 patient presented with peripheral vascular disease. This is in accordance with the impression that normal tension glaucoma is an ocular manifestation of widespread vascular disease.

2 patients had chronic headache – migraine. This may be explained by the fact that both the pathologies have vasospasm in common. Previous studies also have found that migraine is common among patients with normal tension glaucoma than among other types of glaucomas.

7. OPTIC NERVE HEAD CHANGES

CUP: DISC RATIO DISTRIBUTION

On fundoscopic examination, 70 eyes(35%) presented with C:D ratio of 0.7, 63 eyes(31%) had C:D ratio of 0.6 , 40 eyes(20%) had C:D ratio of 0.5, 18 eyes(9%) had C:D ratio of 0.8 and 6 eyes(3%) had C:D ratio of 0.4.



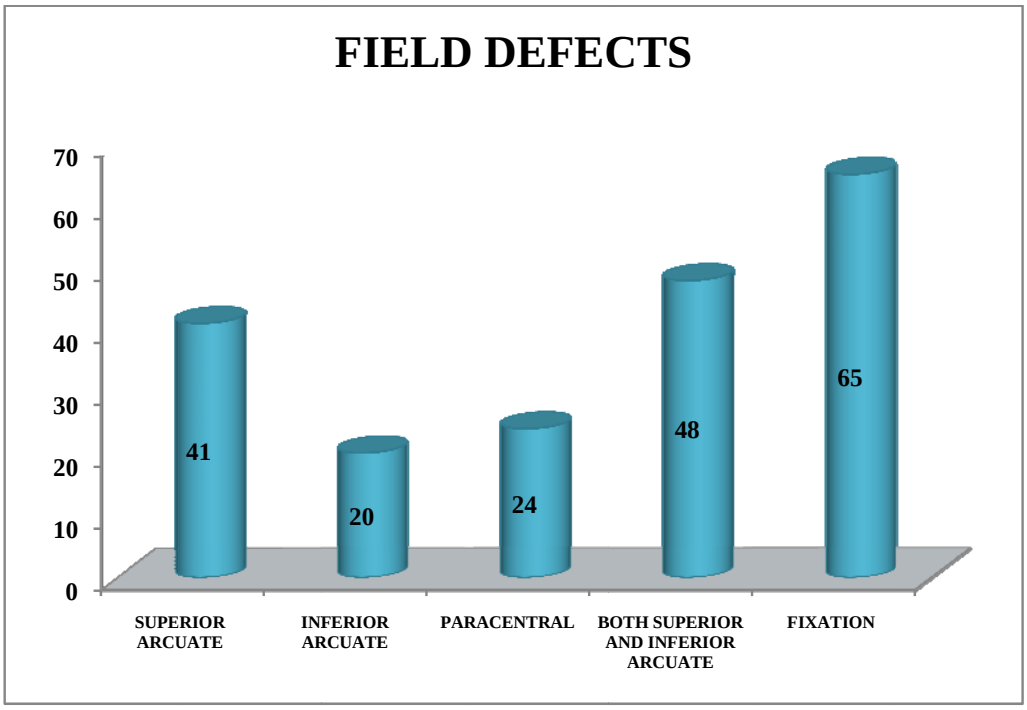
FUNDUS FINDINGS	NO OF EYES	PERCENTAGE
NASALISATION OF VESSELS	158	79%
BAYONETTING OF VESSELS	98	49%
LAMINAR DOT SIGN	146	73%
BARING OF CIRCUMLINEAR VESSELS	30	15%
SPLINTER HEMORRHAGE	9	4%
PERIPAPILLARY ATROPHY	82	41%

8. FIELD DEFECTS DISTRIBUTION

65 out of 198 eyes (32%) had field defects involving fixation area. In larger proportion of patients with normal tension glaucoma visual field defects closer to fixation occur.[1,39]

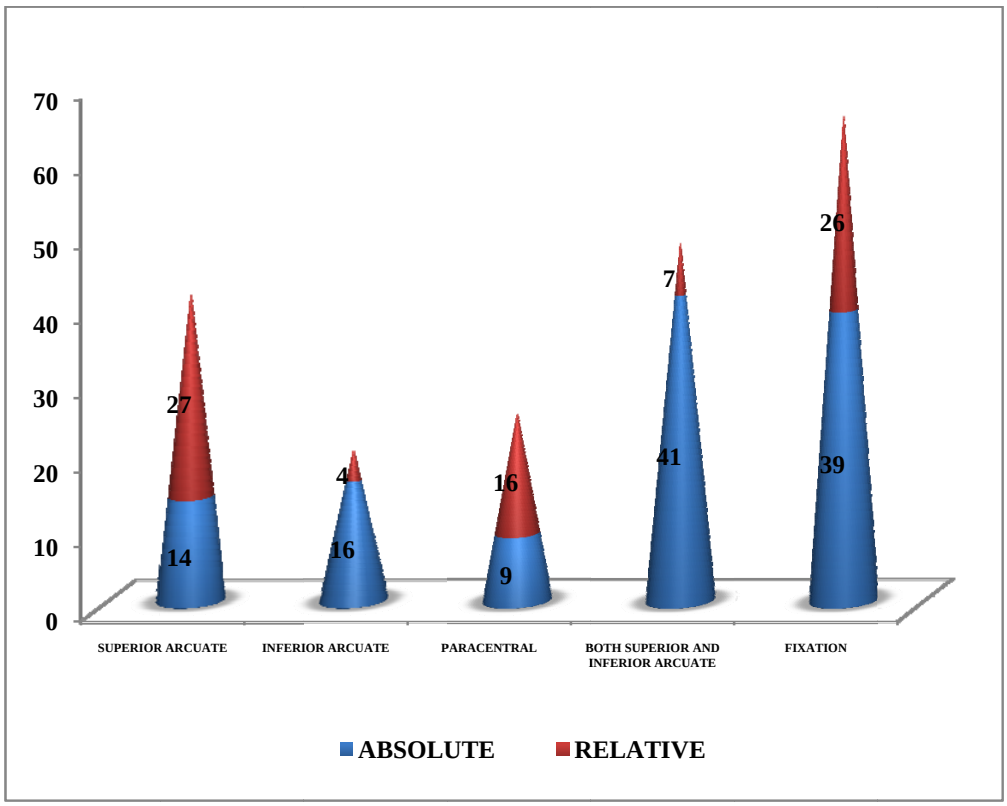
48 eyes(24%) had both superior arcuate and inferior arcuate defects. This is because of the fact that the disease is asymptomatic and it is detected usually while the patients come for check up for defective vision.

Superior arcuate defects(41 eyes) (20%)are more than the inferior arcuate defects(20 eyes)(10%), as in all types of primary open angle glaucoma.[1]

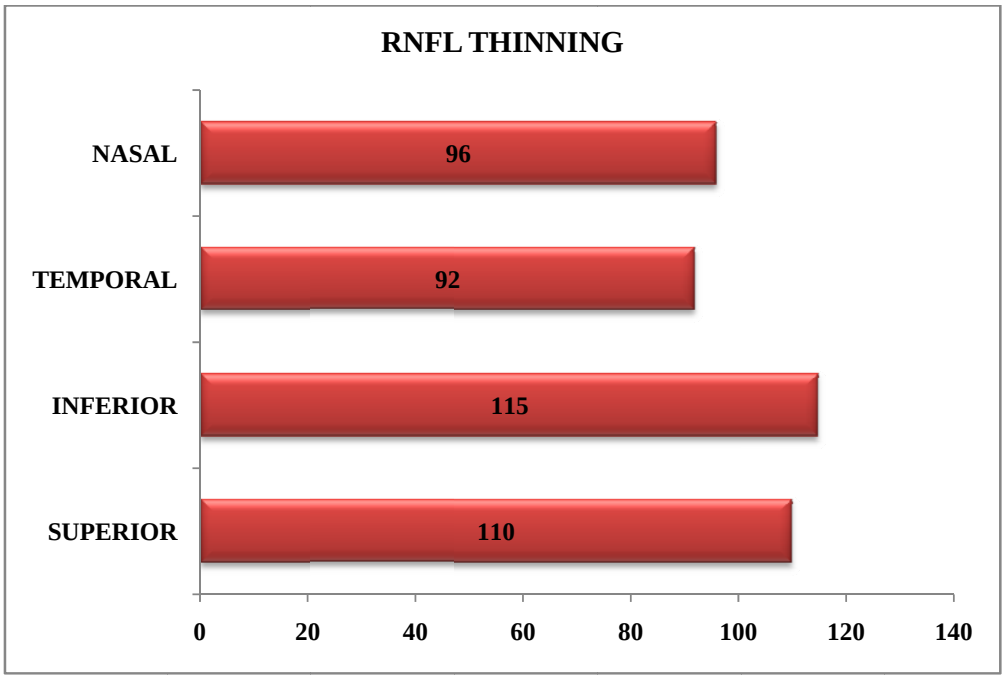


9. ABSOLUTE AND RELATIVE DEFECTS

The absolute scotomas were higher than the relative scotomas in all areas of glaucomatous visual field loss in the series. This might be because of the fact that patients donot present early in case of glaucoma as the disease process is asymptomatic.



10. RETINAL NERVE FIBER LAYER THINNING DISTRIBUTION

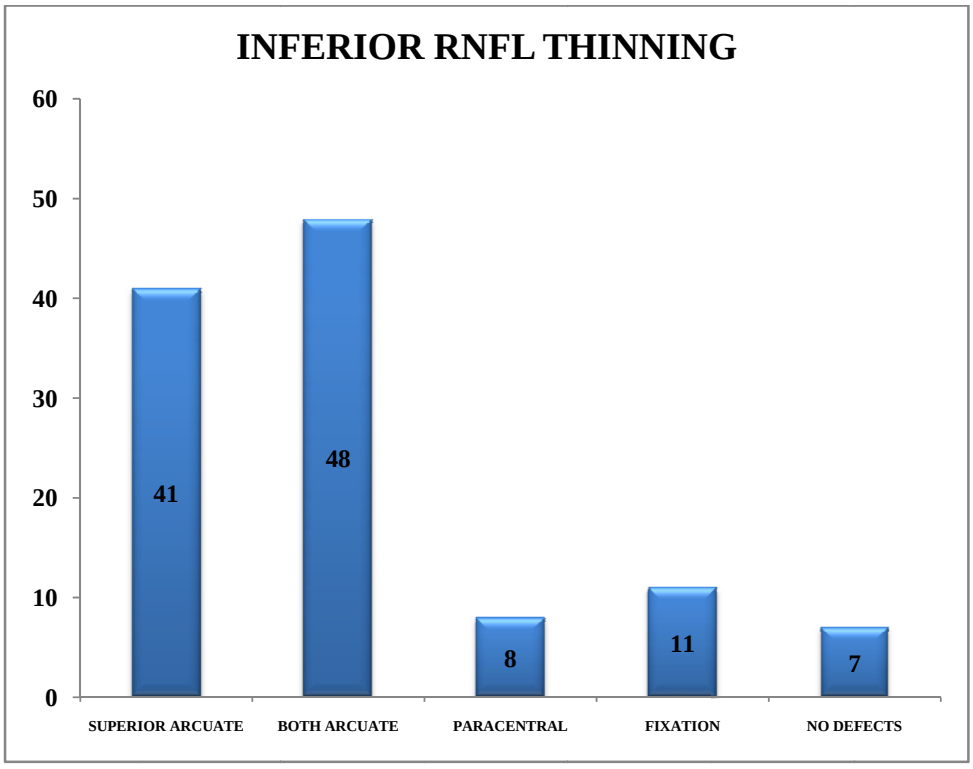


The retinal nerve fiber layer thinning is obtained on comparison with the age wise and quadrant wise normal values. The retinal nerve fiber layer thickness in the normal population is calculated by the computerized Optical coherence tomography and stored as it's normative database. In the study, maximum number of eyes(112eyes)(56%) presented with inferior RNFL thinning, followed by superior thinning in110eyes(55%) , nasal thinning in 96eyes(48%) and temporal thinning in 92eyes(46%). This is in accordance with the thinning pattern distribution in all primary open angle glaucomas including Normal tension glaucoma.[1]

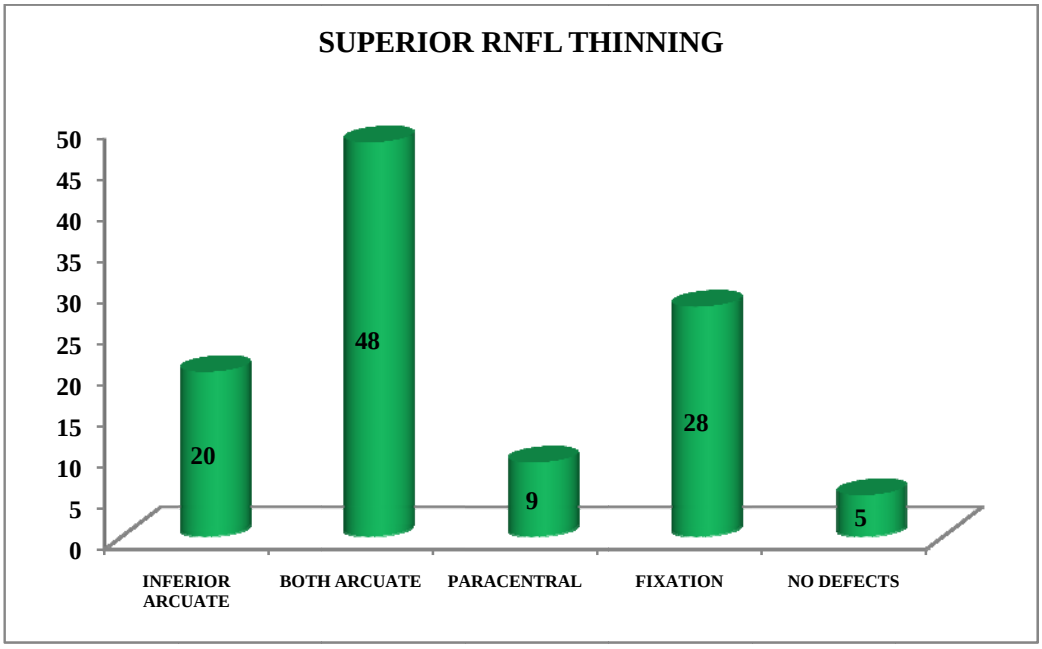
11. CORRELATION OF RNFL THICKNESS WITH THE FIELD DEFECTS

INFERIOR RNFL THINNING

Of the 115 eyes with inferior RNFL thinning, 41eyes had superior arcuate scotoma, 48 eyes had both arcuate scotoma, 8 eyes had paracentral defects, 11 eyes had fixation scotoma and 7eyes had no defects.

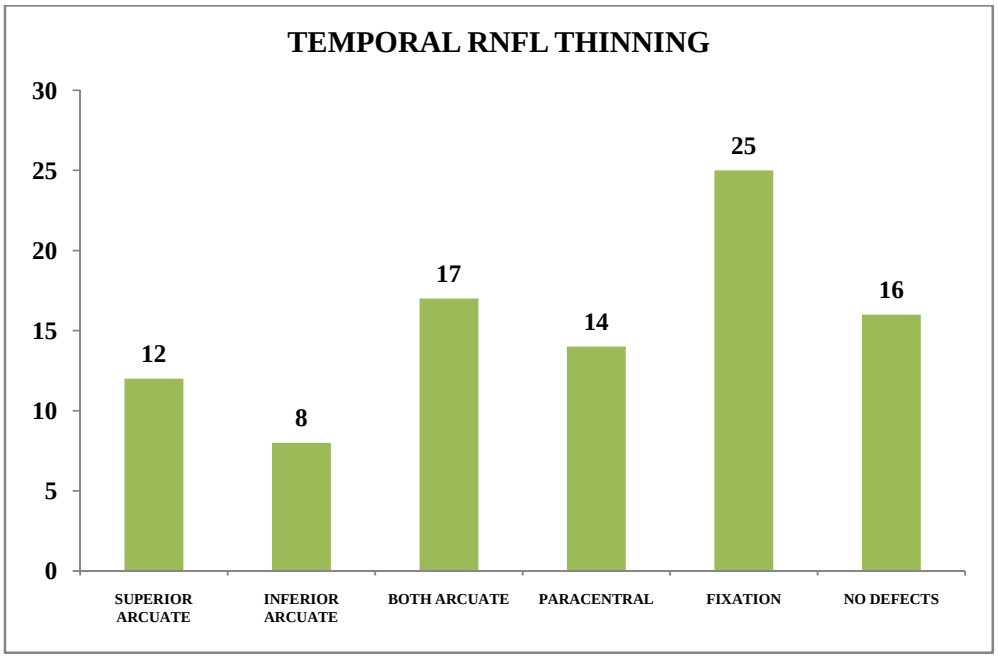


SUPERIOR RNFL THINNING



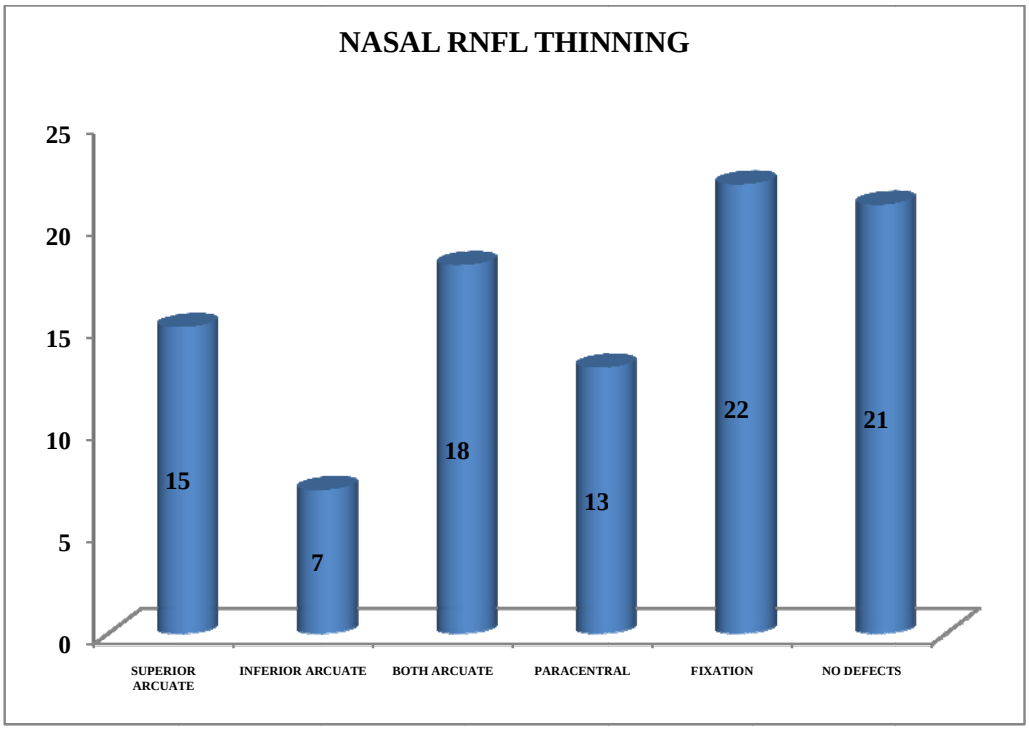
Out of the 110 eyes with superior RNFL thinning, 48 eyes had both arcuate scotoma and 20 eyes had inferior arcuate scotoma. 5 eyes had no field defects.

TEMPORAL RNFL THINNING



Out of the 92 eyes with temporal retinal nerve fiber layer thinning, 12 eyes had superior arcuate area defects, 8 eyes had inferior arcuate area defects, 17 eyes had both arcuate and 14 eyes had paracentral defects. 25 eyes had fixation area defects whereas 16 eyes had no field defects.

NASAL RNFL THINNING

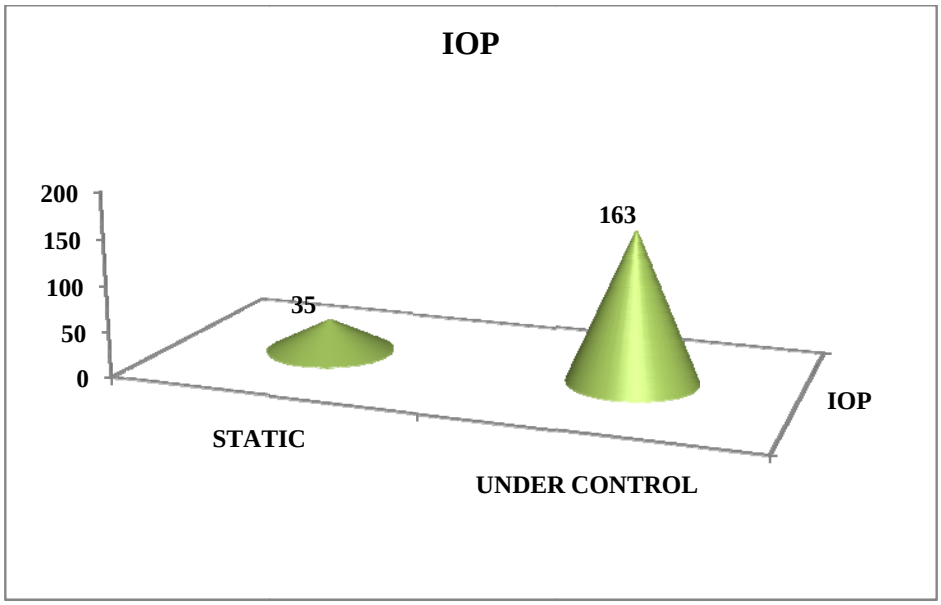


Out of the 96 eyes with nasal RNFL thinning, 22 eyes had fixation area defects . 21 eyes had no defects.

Combining all quadrants , 53 eyes(26%) had retinal nerve fiber layer thinning but no field defects. This indicates that field defects might occur in these patients if not treated. Thus, OCT helps in diagnosing glaucoma at an early stage.

12. FOLLOW UP : FIRST VISIT

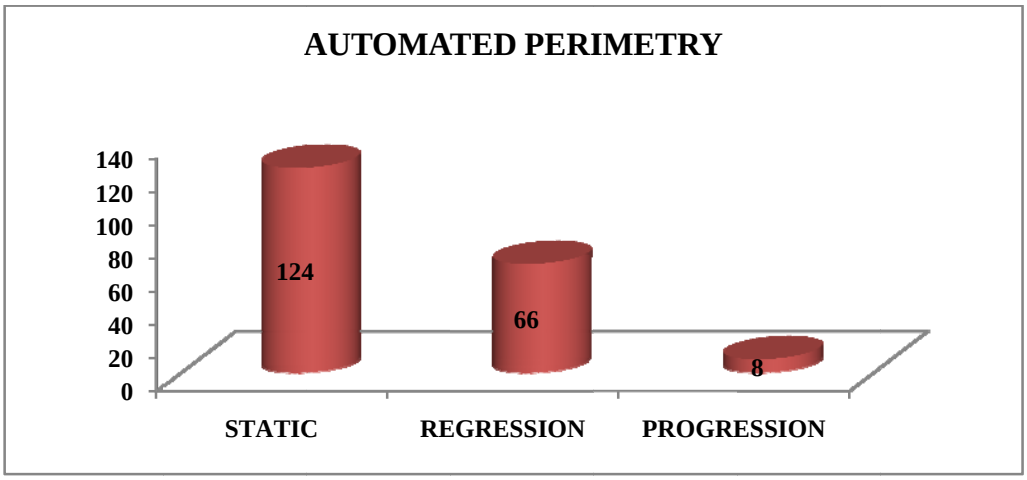
INTRAOCULAR PRESSURE CHANGES



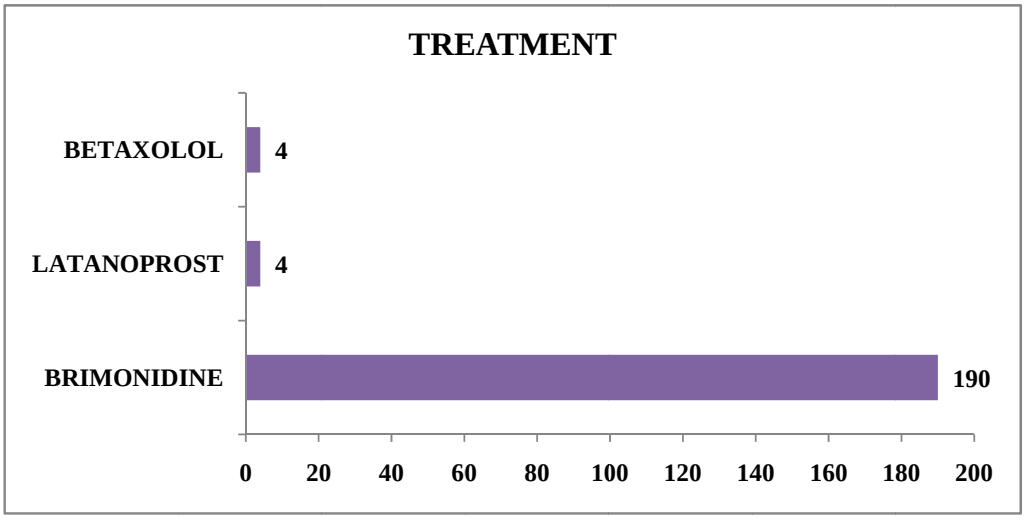
The intraocular pressure reduction of 30 percent was achieved in 163 eyes(82%) who were on treatment with brimonidine eye drops twice a day. The compliance was good in 75 patients and fair in 25 patients.

The fundus changes remained static in all the 198 eyes(100%).

CHANGES IN THE FIELD DEFECTS



TREATMENT

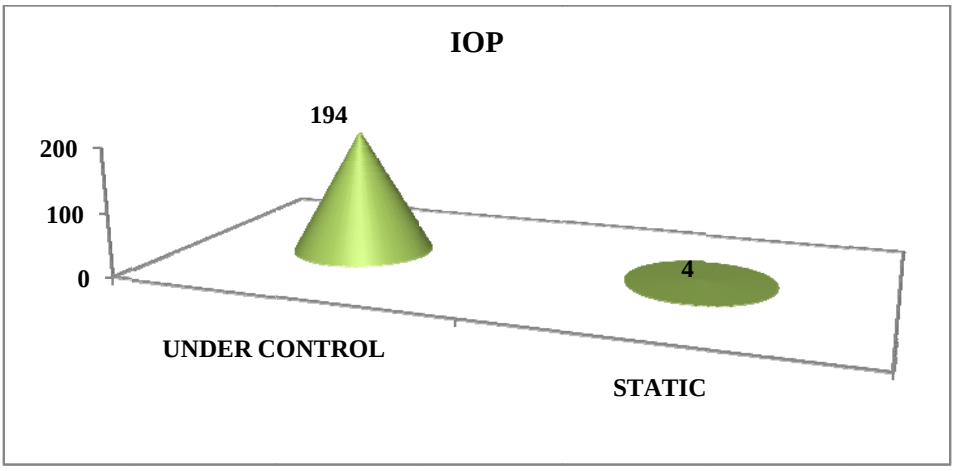


The field changes were static in 124 eyes(62%), as they were absolute defects. The field defects showed regression in 66 eyes(33%) and progression in 8 eyes(4%).

The treatment was changed to latanoprost eye drops once a day in 4 eyes and betaxolol eye drops twice a day in 4 eyes.

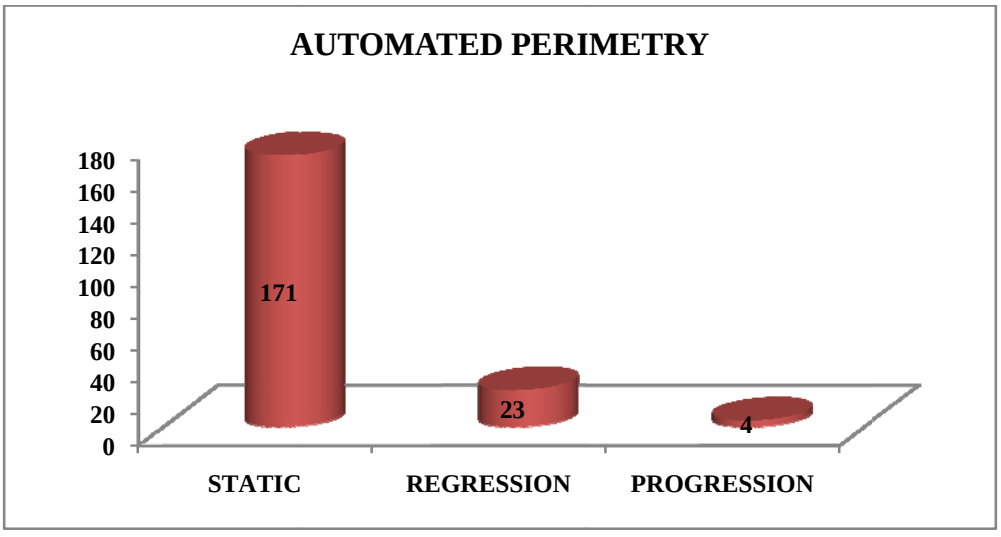
13. FOLLOW UP: SECOND VISIT

INTRAOCULAR PRESSURE CHANGES

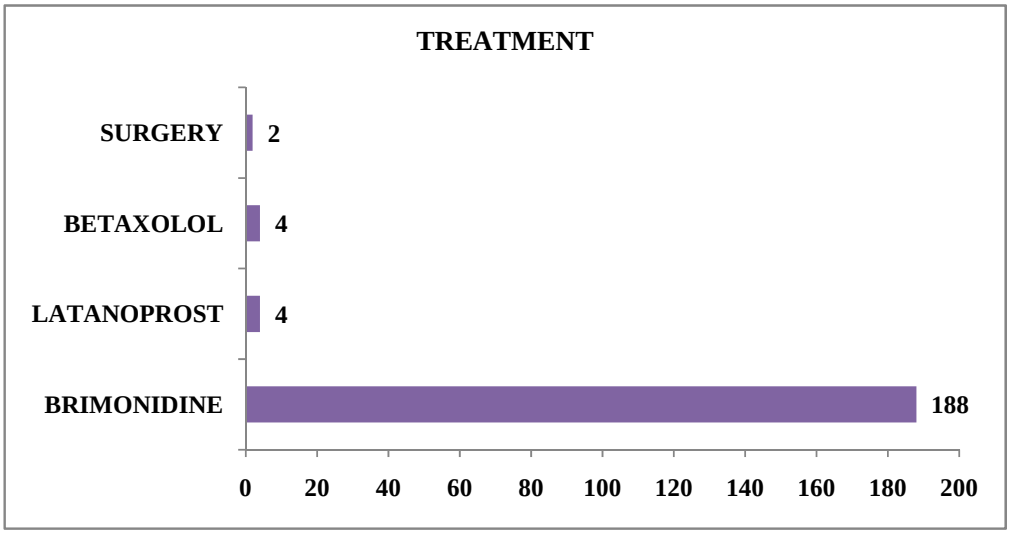


FUNDUS CHANGES: were static in 196 eyes and progressed in 4 eyes.

FIELD CHANGES



TREATMENT



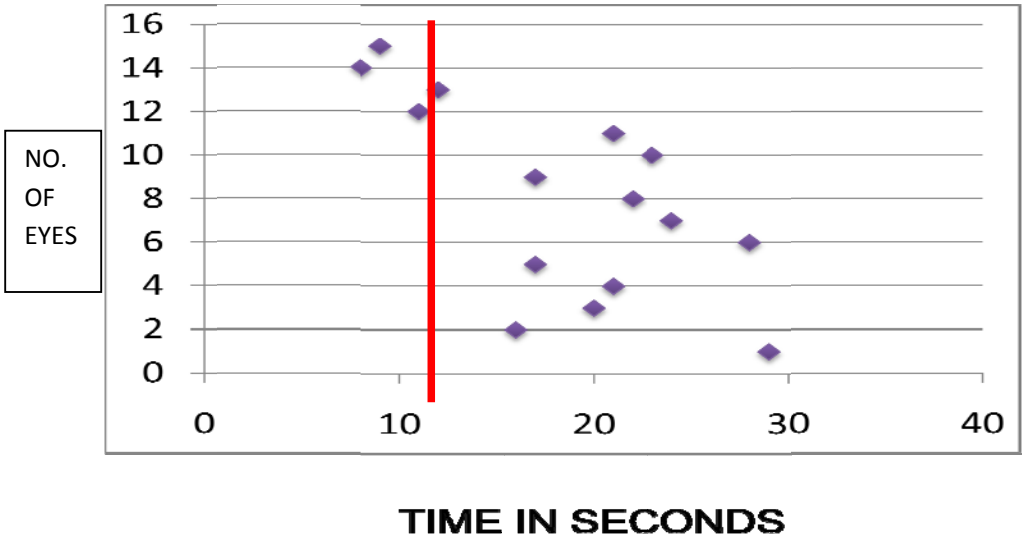
The intraocular pressure was brought under control in 194 eyes(97%) and was still static in 4 eyes(2%). The fundus changes were static in 196 eyes (98%) and were progressive in 2 eyes(1%). The field changes were static in 171 eyes(86%) on comparison with the previous fields and 23 eyes(11%) showed regression of field defects and 4 eyes(2%) showed progression.

The treatment was changed to latanoprost in 2 eyes which showed progression. In the other 2 eyes, Brimonidine + Timolol Combination eye drops twice a day was given for one month. The intraocular pressure remained static, so surgery (trabeculectomy) was done to prevent further progression, since they had defective vision in the other eye. On post operative follow up the intraocular pressure was consistently at 8 mm of Hg and 10 mm of Hg in both the eyes respectively, thus bringing down the intraocular pressure by 30 % in both the eyes.

14. FUNDUS FLUORESCEIN ANGIOGRAPHY OF THE RETINA AND OPTIC NERVE HEAD

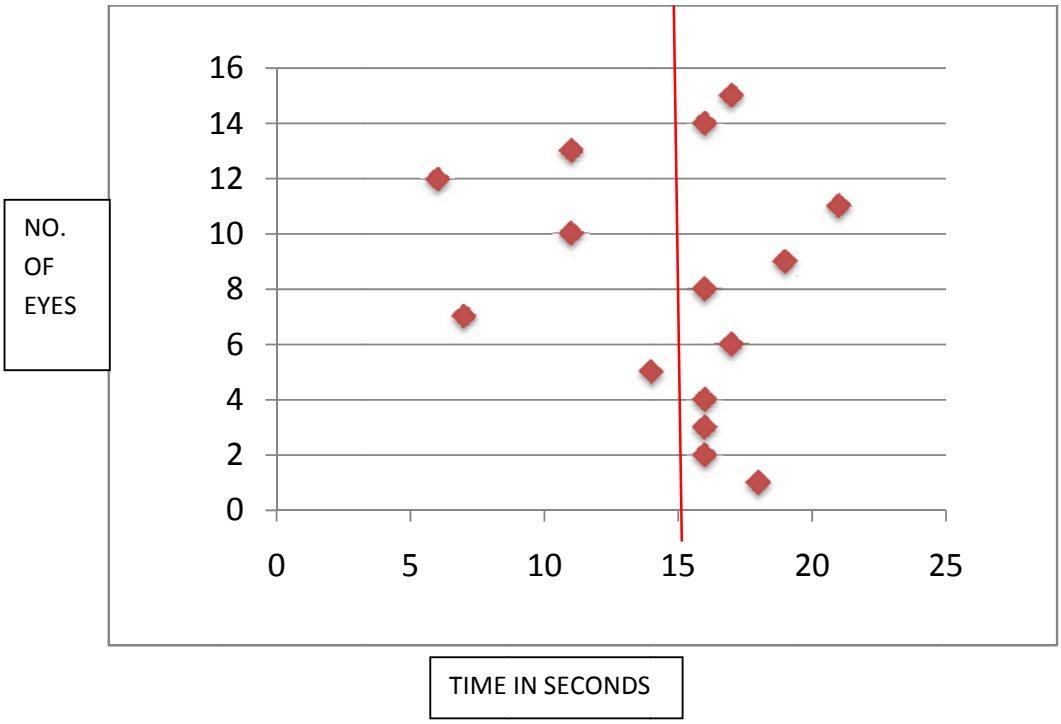
Fundus flourescein angiography was done in 15 patients and the arm-choroidal filling time, arteriovenous transit time and the filling defects on the optic neve head were noted. The filling defects on the optic nerve head were correlated with the field defects as recorded by the automated perimetry-(Octopus 301- G1x program- TOP Strategy).

ARM- CHOROIDAL FILLING TIME



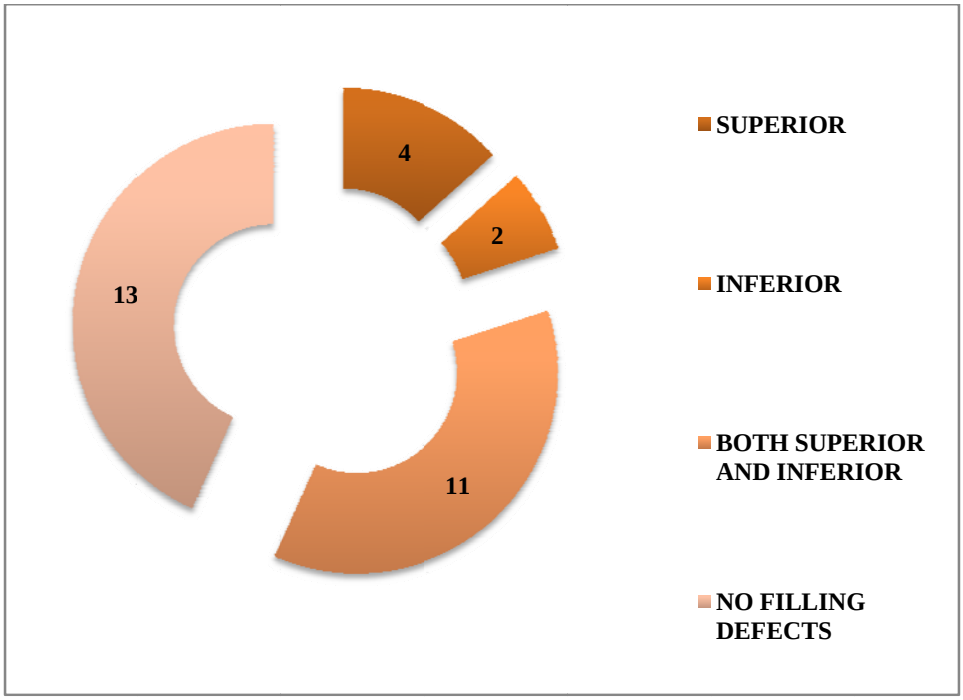
The normal arm choroidal filling time in less than 50 years of age is 10-12 seconds and in more than 50 years of age it is 12-15 seconds.[30] In our study, the arm choroidal filling time was delayed in 11 out of 15 patients.

ARTERIO- VENOUS TRANSIT TIME



The normal arterio-venous transit phase time is 10-12 seconds and above 15 seconds is considered abnormal.[30] In our study, 10 out of 15 patients had prolonged arterio-venous transit time. In patients with normal tension glaucoma , retinal arterio venous passage times are prolonged possibly from the increased resistance in the central retinal and posterior ciliary arteries.

FILLING DEFECTS ON THEOPTIC NERVE HEAD DISTRIBUTION



Sector shaped hypofluorescence refers to a wedge shaped hypofluorescence extending from the edge of the neuroretinal rim towards the center of the disc involving the adjacent part of the optic cup. This is usually seen in normal tension glaucoma. These focal defects occur primarily in the inferior and superior poles of the optic nerve head.[1,25] In our study 17 out of 30 eyes had filling defects. Most of the eyes (11) had both superior and inferior filling defects.

CORRELATION OF FILLING DEFECTS ON ONH WITH THE FIELD DEFECTS

NO. OF EYES	AREA OF SCOTOMA	FILLING DEFECTS
2 /2	SUPERIOR ARCUATE	INFERIOR
3/4	INFERIOR ARCUATE	SUPERIOR
8/11	BOTH ARCUATE	BOTH SUPERIOR AND INFERIOR

The filling defects on the optic nerve head were compared and correlated with the field defects. Most of the eyes (13 /17 eyes) had corresponding scotomas either in the superior arcuate area or inferior arcuate area or both. Since the areas of absolute filling defects indicate persisting hypoperfusion, they are said to correlate with visual field loss.

RESULTS

- The commonest age group(68%) in the study was between 41-60 years.
- Males (61%) were predominant than the females (39%) in the study group.
- 171 eyes (86%) presented with visual acuity of 6/36 and above. 25 eyes (12%) presented with visual acuity below 6/36. 132 eyes (66%) had refractive error, 53 eyes(26%) had early lens changes and 12 eyes (6%) had posterior capsular opacification.
- At presentation, 125 eyes (63%) had intraocular pressure in low teens and mid teens. 69 eyes (34%) had intraocular pressure in high teens.
- 106 eyes (53%) had normal central corneal thickness(CCT) , 82 eyes (41%) had higher CCT values and 10 eyes (5%) had lower CCT values.
- 51 patients(51%) had one of the associated risk factors like hypertension, diabetes, hypotension, acute blood loss, hemorrhoids, ischemic heart disease, migraine and hypercholesterolemia.

- 133 eyes (67%) had cup disc ratio distribution between 0.6 and 0.7. 158 eyes (79%) had nasalization of vessels, 98 eyes (49%) had bayoneting of vessels, 146 eyes (73%) had laminar dot sign, 30 eyes (15%) had baring of circumciliary vessels , 9 eyes (4%) had splinter hemorrhage and 82 eyes (41%) had peripapillary atrophy.
- 65 eyes(32%) had field defects involving fixation area and 48 eyes (24%) had both arcuate scotomas.
- 112 eyes (56%) had inferior retinal nerve fiber layer(RNFL) thinning, 110 eyes(55%) had superior RNFL thinning, 96 eyes(48%) had nasal RNFL thinning and 92 eyes (46%) had temporal RNFL thinning.
- 53 eyes (26%) had retinal nerve fiber layer thinning in Optical Coherence Tomography ,but no field defects in automated perimetry.
- On first followup (after 3 months), Intraocular pressure reduction was achieved in 163 eyes(82%) on treatment with topical brimonidine. Fundus changes remained static in all eyes. Field defects were static in 124 eyes (62%), regressive in 66 eyes (33%) and progressive in 8 eyes (4%). Latanoprost

was substituted in 4 eyes and Betaxolol was substituted in 4 eyes.

- On the subsequent follow up (after 6 months), Intraocular pressure reduction was achieved in 194 eyes(97%) .Fundus changes were progressive in 2 eyes (1%). Field defects were progressive in 4 eyes(2%). 2 eyes were started on topical latanoprost and in two eyes , trabeculectomy was done with post operative intraocular pressure reduction of 30 % and static field defects on follow up.
- On Fundus Flourescein angiographic studies, arm choroidal filling time was delayed in 11 out of 15 eyes and arterio-venous transit time was prolonged in 10 out of 15 eyes.
- 17 out of 30 eyes had filling defects on the optic nerve head of which 13 eyes had corresponding field defects.

DISCUSSION

Normal tension glaucoma is a diagnostic conundrum[1] because a diagnosis is made only after diurnal variation of intraocular pressure is done and no recording goes above 21 mm of Hg and after correcting the intraocular pressure for central corneal thickness. Thus central corneal thickness measurement plays an important role in the diagnosis of this disease entity.

Fundus examination with +90 D biomicroscopy is a must in all refraction cases because the disease as such is asymptomatic till the field defects progress. So, detecting patients early and beginning treatment prevents field defects and blindness due to this disease.

Since the disease presents in the elderly especially in the 5^{th} to 6^{th} decade, annual or two yearly routine fundus examination leads to early detection and hence early treatment.

The presence of associated risk factors suggests that the vascular pathology either by causing vasospasm or by hypoperfusion might affect the optic nerve head resulting in the progression or the establishment of the disease process.

Optical coherence tomography helps in detecting the retinal nerve fiber layer thinning even before the occurrence of the field defects. Thereby OCT helps in early diagnosis of normal tension glaucoma.

The response to topical brimonidine is good in these set of patients and the other drugs which can also be used are latanoprost and betaxolol. Thus, medical control of intraocular pressure (reduction of IOP by 30%) is the main modality of treatment in normal tension glaucoma. The role of surgery is minimal in case of normal tension glaucoma, but post surgical reduction of intraocular pressure below 30% arrests further progression of the defects.

Delayed choroidal filling time and prolonged arterio-venous transit time indicate that hypoperfusion might be an etiological factor in normotensive glaucoma. This opens an eye for further research in the field of Normal tension glaucoma, where the etiology is still obscure. In addition to it Doppler flowmetry studies might throw a light on the newer modalities of management of Normal tension glaucoma.

CONCLUSION

Central corneal thickness measurement and Diurnal variation test play an important role in the diagnosis of Normal tension glaucoma.

Optical Coherence Tomography aids in the early detection of structural damage in the form of retinal nerve fiber layer thinning even before it is evident as field defects.

The progression of field defects in Normal tension glaucoma is arrested by 30% reduction of intraocular pressure, which is achieved by medical management with topical brimonidine in 95 % of the eyes.

The presence of associated risk factors and fundus fluorescein angiographic studies indicate that hypoperfusion might be an etiological factor in Normal tension glaucoma.

LIST OF SURGERIES PERFORMED

S. No	NAME	AGE / SEX	IP. no.	DIAGNOSIS	DATE OF SURGERY	SURGERY PERFORMED
1.	SAROJA	55/F	416107	LE-MC	25.7.07	LE-ECCE WITH PCIOL
2.	RAJI	60/F	418191	BE-IMC	19.9.07	RE-ECCE WITH PCIOL
3.	ELUMALAI	60/M	418270	BE-IMC	20.3.08	LE-ECCE WITH PCIOL
4.	RAJESHWARI	60/F	426798	BE-IMC	30.5.08	LE-SICS WITH PCIOL
5.	RANI	62/F	426021	BE-IMC	6.6.08	RE-SICS WITH PCIOL
6.	KUPPU	70/F	422082	BE-IMC	3.3.09	RE-SICS WITH PCIOL
7.	SUSEELA	62/F	461141	RE-IMC	14.4.09	RE-SICS WITH PCIOL
8.	JOSEPH	60/M	431327	BE-IMC	27.5.08	LE-SICS WITH PCIOL
9.	KALAVATHY	45/F	4222021	RE-NVG	19.8.09	RE-TRABECULECTOMY
10	PONNAMMA	50/F	4322451	RE-CHR.DAC	18.9.09	RE-DCR
11	MALATHY	45/F	4344273	LE-CHR.DAC	9.10.09	LE-DCR
12	PUSHPA	43/F	4314523	RE-PTERYG.	17.6.09	RE-PTERYG. EXCISION WITH AMNIOTIC MEMBRANE GRAFT
13	MOHAMED	46/M	4423122	LE- PTERYG.	15.7.09	LE-PTERYG.EXCISION WITH CONJ.AUTOGRAFT
14	MANJULA	48/F	4432761	RE-PTERYG.	5.8.09	RE-PTERYG.EXCISION WITH AMNIOTIC MEMBRANE GRAFT
15	MALLIKA	50/F	432156	RE-IMC	10.8.09	RE-SICS WITH PCIOL
16	SRINIVASAN	60/M	432257	RE-PSC	24.8.09	RE-SICS WITH PCIOL
17	DHANAPAL	56/M	421343	RE-ENTROPION	18.9.09	RE-LATERAL TARSAL STRIP PROCEDURE
18	KUPAYEE	68/F	427833	RE-CHR.DAC	25.9.09	RE-DACRYOCYSTECTOMY
19	KUMARESAN	65/M	423564	LE-CORNEAL TEAR	16.10.09	LE- CORNEAL TEAR SUTURING DONE
20	MURUGAVEL	50/M	423534	RE-IMC	14.10.09	RE-SICS WITH PCIOL

S. No	NAME	AGE / SEX	IP. no.	DIAGNOSIS	DATE OF SURGERY	SURGERY PERFORMED
21	KANDASAMY	53/M	423345	LE-IMC	21.10.09	LE-SICS WITH PCIOL
22	VELAYAN	50/M	423546	RE-PSC	28.10.09	RE-SICS WITH PCIOL
23	SUBAIYAN	58/M	426754	RE-MC	2.11.09	RE-SICS WITH PCIOL
24	KUPPUSAMY	60/M	426765	LE-PSC	9.11.09	LE-SICS WITH PCIOL
25	MUKILAN	50/M	425656	RE-IMC	25.11.09	RE-SICS WITH PCIOL

ABBREVIATIONS

1. RE/LE - RIGHT EYE / LEFT EYE
2. IMC - IMMATURE CATARACT
3. MC - MATURE CATARACT
4. PSC - POSTERIOR SUBCAPSULAR CATARACT
5. CHR.DAC - CHRONIC DACRYOCYSTITIS
6. PTERYG - PTERYGIUM
7. ECCE - EXTRACAPSULAR CATARACT EXTRACTION
8. SICS – SMALL INCISION CATARACT SURGERY
9. PCIOL - POSTERIOR CHAMBER INTRAOCULAR LENS
10. DCR - DACRYOCYSTORHINOSTOMY

NO	NAME	AGE	SEX	EYE	V/A	CCT	CIOP	C:D	AP	RF	TRT	SUP	INF	TEM	NAS	P1	C:D	AP	TRT	P2	C:D	AP	TRT	HB %	LP
1	PALANI	48	M	RE	6/12 PH6/9	578	14	0.7	A/S /I	HT	B	107	110	54	90	12	0.7	A /S I	B	12	0.7	A/ S I	B	N	N
				LE	6/12 PH 6/6	602	17	0.8	A / I		B	97	120	56	81	14	0.8	A/ I	B	12	0.8	A PC	B		
2	VINAYAK	59	M	RE	6/36 PH 6/6	522	15	0.5	R/I	-	B	121	130	96	110	14	0.5	R/ PC	B	14	0.5	R/ PC	B	N	N
				LE	6/18 PH 6/6	535	16	0.5	-		B	122	132	90	102	14	0.5	-	B	14	0.5	-	B		
3	KRISHNAMOORTHY	48	M	RE	6/60 PH 6/6	520	14	0.6	A/S/F	-	B	126	139	82	86	12	0.6	R / S /I /F	B	12	0.6	R/ F	B	N	N
				LE	6/12 PH 6/9	522	14	0.6	A/F		B	123	156	67	114	14	0.6	A/F	B	12	0.6	A/F	B		
4	KARPAGAM	55	F	RE	6/60 PH 6/18	553	15	0.5	A /S/I	MG	B	129	146	69	135	12	0.5	R/ S/I	B	12	0.5	R/S/I	B	N	N
				LE	6/36 PH 6/6	550	14	0.7	R/PC		B	130	112	79	106	14	0.7	R/ S/I	BL	14	0.7	R/S/I	BL		
5	RAJENDRAN	58	M	RE	6/36 PH 6/9	578	15	0.7	A / S/ I	HC	B	138	119	55	88	14	0.7	A/ S I	B	14	0.7	A/ S I	B	N	ABN
				LE	6/24 PH 6/9	580	15	0.7	A/ I		B	168	86	68	121	12	0.7	A /I	B	14	0.7	A / I	B		
6	RANGANAYAKI	65	F	RE	6/12 PH 6/6	534	12	0.7	A/ PC/F	HT	B	125	152	63	119	12	0.7	A/ F	B	12	0.7	A/ F	B	N	N
				LE	6/24 PH 6/6	540	12	0.6	A/ F		B	149	111	106	80	12	0.6	A/ F	B	12	0.6	A/F	B		
7	MONISHA	35	F	RE	6/36 PH 6/6	580	15	0.4	A / I	-	B	109	143	120	60	14	0.4	A/ I	B	14	0.4	A/I	B	N	N
				LE	6/24 PH 6/6	582	15	0.6	A/ I /F		B	150	154	79	84	12	0.6	A/F	B	14	0.6	A/F	B		
8	VIJAYAKUMAR	37	M	RE	6/24 PH 6/6	522	16	0.7	R/ S	-	B	112	138	100	83	12	0.7	R/PC	B	14	0.7	R/PC	B	N	N
				LE	6/9 PH 6/6	529	14	0.8	A/S/I		B	88	113	57	90	14	0.8	A/S/I	B	14	0.8	A/S/I	B		
9	NARGEES	44	F	RE	6/9 PH 6/6	460	18	0.7	A/S	HOT	B	82	84	63	90	16	0.7	A/S	B	16	0.7	A/S	B	N	N
				LE	6/9 ph 6/6	458	16	0.8	A/S/F		B	123	156	67	114	16	0.8	A/S/F	B	14	0.8	A/S/F	B		
10	LALITHA	55	F	RE	6/12 PH 6/6	583	15	0.5	R/S	-	B	133	147	76	111	14	0.5	R/S	B	14	0.5	R/S/F	L	N	N
				LE	6/24 PH 6/9	587	15	0.7	A/S/F		B	81	69	63	54	14	0.7	A/S/F	B	14	0.7	A/S	B		
11	DAYAL	60	M	RE	6/12 PH 6/6	530	14	0.7	A/ S	HM	B	107	120	67	117	12	0.7	A/S	B	14	0.7	A/S	B	DEC	N
				LE	6/24 PH 6/6	527	14	0.6	A/ S/F		B	122	154	75	113	12	0.6	A/S	B	14	0.6	A/S	B		
12	SADAGOPAN	65	M	RE	6/12 PH 6/9	535	14	0.6	R/S/I	HT	B	136	148	80	100	12	0.6	R/S	B	12	0.6	R/S	B	N	N
				LE	6/12 PH 6/6	530	14	0.7	A/F		B	156	152	72	101	14	0.7	A/ F	B	12	0.7	A/F	B		
13	KANNIAMMA	60	F	RE	6/36 PH 6/12	540	13	0.5	R/PC	-	B	136	140	64	102	10	0.5	R/PC	B	12	0.5	R/PC	B	N	N
				LE	6/24 PH 6/9	538	12	0.6	R/PC/F		B	136	143	102	100	12	0.6	R/PC	B	12	0.6	R/PC	B		
14	SHANTHA	50	F	RE	6/12 PH 6/6	530	16	0.8	A/S/I	-	B	121	121	62	88	16	0.8	A/S/I	B	14	0.8	A/S/I	B	N	N
				LE	6/24 PH 6/6	529	16	0.7	A/S/F		B	139	80	81	105	14	0.7	A/S/F	B	14	0.7	A/S/F	B		
15	SUBRAMANI	50	M	RE	6/24 PH 6/9	540	14	0.6	R/S	IHD	B	137	147	100	80	12	0.6	R/PC	B	14	0.6	R/PC	B	N	N
				LE	6/12 PH 6/6	535	15	0.5	R/S		B	145	156	79	93	12	0.5	R/F	B	14	0.5	R/F	B		
16	PALANINATHAN	41	M	RE	6/24 PH 6/6	537	14	0.5	R/F	-	B	163	154	94	115	12	0.5	R/PC	B	14	0.5	R/PC	B	N	N
				LE	6/12 PH 6/6	536	14	0.6	R/S		B	136	142	52	116	12	0.6	R/S	B	14	0.6	R/S	B		
17	RAMAKRISHNAN	65	M	RE	6/12 PH6/9	567	15	0.7	A/S /I	HOT	B	146	158	73	107	12	0.7	A /S I	B	12	0.7	A/ S I	B	N	N

				LE	6/12 PH 6/6	563	16	0.8	A / I		B	125	131	74	83	14	0.8	A/ I	B	12	0.8	A/PC	B		
18	VENKTACHALAM	51	M	RE	6/36 PH 6/6/	522	18	0.5	R/I	-	B	133	159	97	76	14	0.5	R/ PC	B	14	0.5	R/ PC	B	N	N
				LE	6/18 PH 6/6	521	20	0.5	R/S		B	145	134	90	114	18	0.5	R/S	B	18	0.5	-	B		
19	NOOR MOHAMED	65	M	RE	6/60 PH 6/6	520	16	0.6	A/S/F	HOT	B	134	139	75	98	12	0.6	R / S /I /F	B	12	0.6	R/ F	B	N	N
				LE	6/12 PH 6/9	522	14	0.6	A/F		B	123	148	63	109	14	0.6	A/F	B	12	0.6	A/F	B		
20	NOORISLAM	54	M	RE	6/60 PH 6/18	553	15	0.5	A /S/I		B	123	138	56	107	12	0.5	R/ S/I	B	12	0.5	R/S/I	B	N	N
				LE	6/36 PH 6/6/	550	14	0.7	R/PC		B	110	118	69	94	14	0.7	R/ S/I	BL	14	0.7	R/S/I	BL		
21	PARVEEN	29	F	RE	6/36 PH 6/9	538	14	0.7	A / S/ I	HC	B	124	130	74	80	14	0.7	A/ S I	B	14	0.7	A/ S I	B	N	ABN
				LE	6/24 PH 6/9	534	14	0.7	A/ I		B	100	120	76	85	12	0.7	A /I	B	14	0.7	A / I	B		
22	RADHA	48	F	RE	6/12 PH 6/6	521	12	0.7	A/ PC/F	HT	B	121	110	56	80	12	0.7	A/ F	B	12	0.7	A/ F	B	N	N
				LE	6/24 PH 6/6	514	12	0.6	A/ F		B	131	129	88	82	12	0.6	A/ F	B	12	0.6	A/F	B		
23	RAMUTHAI	42	F	RE	6/36 PH 6/6	543	15	0.4	A / I	-	B	161	136	68	90	15	0.4	A/ I	B	14	0.4	A/I	B	N	N
				LE	6/24 PH 6/6	549	14	0.6	A/ I /F		B	134	91	96	116	12	0.6	A/F	B	14	0.6	A/F	B		
24	RAMACHANDRAN	52	M	RE	6/24 PH 6/6	487	16	0.7	R/ S	-	B	103	125	76	100	14	0.7	R/PC	B	14	0.7	R/PC	B	N	N
				LE	6/9 PH 6/6	483	14	0.8	A/S/I		B	98	126	74	75	14	0.8	A/S/I	B	14	0.8	A/S/I	B		
25	LENIN	40	M	RE	6/9 PH 6/6	530	19	0.7	A/S	HOT	B	88	77	76	74	16	0.7	A/S	B	16	0.7	A/S	B	N	N
				LE	6/9 PH 6/6	532	20	0.8	A/S/F		B	122	133	64	91	16	0.8	A/S/F	B	14	0.8	A/S/F	B		
26	LAKSHMI	35	F	RE	6/12 PH 6/6	528	16	0.5	R/S	-	B	121	144	59	111	14	0.5	R/S	B	14	0.5	R/S/F	L	N	N
				LE	6/24 PH 6/9	530	14	0.7	A/S/F		B	126	139	82	86	12	0.7	A/S/F	B	14	0.7	A/S	B		
27	NAGARAJ	67	M	RE	6/12 PH 6/6	532	15	0.7	A/ S	-	B	107	102	78	109	12	0.7	A/S	B	14	0.7	A/S	B	N	N
				LE	6/24 PH 6/6	536	14	0.6	A/ S/F		B	109	153	66	125	12	0.6	A/S	B	14	0.6	A/S	B		
28	SAMPATH	70	M	RE	6/12 PH 6/9	526	14	0.6	R/S/I	HT	B	112	87	54	94	12	0.6	R/S	B	12	0.6	R/S	B	N	N
				LE	6/12 PH 6/6	530	14	0.7	A/F		B	161	181	90	90	14	0.7	A/ F	B	12	0.7	A/F	B		
29	PUSHPARAJ	47	M	RE	6/36 PH 6/12	538	13	0.5	R/PC	-	B	142	116	46	117	12	0.5	R/PC	B	12	0.5	R/PC	B	N	N
				LE	6/24 PH 6/9	538	12	0.6	R/PC/F		B	143	137	62	107	12	0.6	R/PC	B	12	0.6	R/PC	B		
30	PHILOMINA	68	F	RE	6/12 PH 6/6	530	16	0.8	A/S/I	-	B	97	111	56	97	16	0.8	A/S/I	B	14	0.8	A/S/I	B	N	N
				LE	6/24 PH 6/6	536	16	0.7	A/S/F		B	100	99	56	85	14	0.7	A/S/F	B	14	0.7	A/S/F	B		
31	PADMANABAN	65	M	RE	6/24 PH 6/9	540	14	0.6	R/S	HM	B	135	134	72	125	12	0.6	R/PC	B	14	0.6	R/S	L	N	N
				LE	6/12 PH 6/6	535	14	0.5	R/S		B	150	118	53	106	12	0.5	R/F	B	14	0.5	R/F	B		
32	THANGARAJ	61	M	RE	6/60 PH 6/24	532	16	0.6	A/I	-	B	130	148	69	105	16	0.6	A/I	B	14	0.6	A/I	B	N	N
				LE	6/60 PH 6/24	540	16	0.8	A/S/I		B	100	123	56	83	14	0.8	A/S/I	B	14	0.8	A/S/I	B		
33	KUMARI	55	F	RE	6/36 PH 6/9	582	15	0.6	R/S	-	B	146	145	65	114	14	0.6	R/S/I	BL	14	0.6	R/S	BL	N	N
				LE	6/12 PH 6/6	589	15	0.5	R/I		B	140	127	76	119	14	0.5	R/I	B	14	0.5	R/I	B		
34	ASHOK	50	M	RE	6/24 PH 6/9	583	17	0.5	R/S/I	PVD	B	136	133	67	101	14	0.5	R/S	B	14	0.5	R/S	B	N	N
				LE	6/12 PH 6/6	580	15	0.6	R/S/F		B	144	117	82	76	14	0.6	R/S	B	14	0.6	R/S	B		

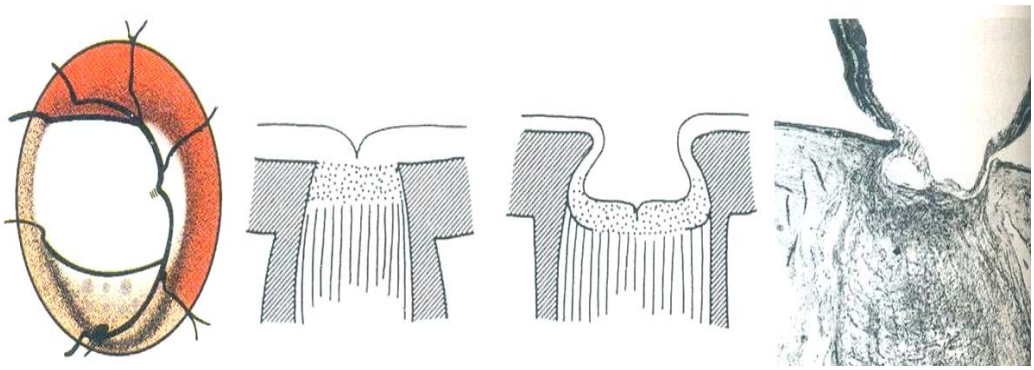
35	BABY	67	F	RE	6/60 PH 6/36	542	14	0.6	A/F	-	B	148	157	61	122	12	0.6	A/F/PC	BL	14	0.6	A/PC	BL	N	N
				LE	6/36 PH 6/24	536	14	0.7	A/S/I		B	159	160	52	95	12	0.7	A/S/I	B	14	0.7	A/S/I	B		
36	LILLY PUSHPAM	64	F	RE	6/60 PH 6/36	530	16	0.7	A/I	DM	B	136	133	67	101	14	0.7	A/I	B	14	0.7	A/I	B	N	N
				LE	6/36 PH 6/24	528	16	0.6	R/ S/I		B	147	141	55	120	14	0.6	R/S	B	14	0.7	R/S	B		
37	JOTHY	63	F	RE	6/9 PH 6/6	532	16	0.8	A/S/F		B	121	110	60	76	16	0.8	A/S/F	B	14	0.8	A/S/F	B	N	N
				LE	6/12 PH 6/6	536	14	0.5	R/S	-	B	171	178	86	139	14	0.5	R/S	B	14	0.5	R/S	B		
38	RAGHUNATHAN	45	M	RE	6/24 PH 6/9	530	14	0.7	A/S/F	MG	B	121	132	64	98	12	0.7	A/S/F	B	14	0.7	A/S	B	N	N
				LE	6/12 PH 6/6	527	14	0.7	A/ S	-	B	128	117	66	86	12	0.7	A/S	B	14	0.7	A/S	B		
39	ESWARAN	60	M	RE	6/24 PH 6/6	536	14	0.6	A/ S/F		B	132	109	63	103	11	0.6	A/S	B	14	0.6	A/S	B	N	N
				LE	6/12 PH 6/9	535	14	0.6	R/S/I	HT	B	109	99	65	76	12	0.6	R/S	B	12	0.6	R/S	B		
40	SRINIVASAN	59	M	RE	6/12 PH 6/6	585	15	0.7	A/F		B	129	116	60	85	14	0.7	A/ F	B	12	0.7	A/F	B	N	N
				LE	6/36 PH 6/12	589	13	0.5	R/PC	-	B	135	132	65	104	12	0.5	R/PC	B	12	0.5	R/PC	B		
41	ROBERT	70	M	RE	6/24 PH 6/9	538	12	0.6	R/PC/F		B	137	132	82	76	12	0.6	R/PC	B	12	0.6	R/PC	B	N	N
				LE	6/12 PH 6/6	530	16	0.8	A/S/I	-	B	108	121	65	54	16	0.8	A/S/I	B	14	0.8	A/S/I	B		
42	PITCHAIAMMAL	60	F	RE	6/24 PH 6/6	536	16	0.7	A/S/F	IHD	B	133	142	79	106	14	0.7	A/S/F	B	14	0.7	A/S/F	B	N	N
				LE	NO PL	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
43	ESWARAPILLAI	57	M	RE	6/60 PH 6/36	540	14	0.6	A/S/I	DM	B	129	146	69	135	11	0.6	A/S/I	B	14	0.6	A/S/I	B	N	N
				LE	6/36 PH 6/18	542	14	0.7	A/S/I		B	119	132	65	98	11	0.7	A/S/I	B	14	0.7	A/S/I	B		
44	RAMAN	55	M	RE	NO PL	-	-	-	-	DM	B	-	-	-	-			-	-	-	-	-	-	N	N
				LE	6/12 PH 6/9	537	14	0.6	A/S		B	126	140	66	132	14	0.6	A/S	B	14	0.6	A/S	B		
45	KASINATHAN	65	M	RE	6/18 PH 6/12	525	16	0.6	A/I	DM	B	133	136	78	104	16	0.6	A/I	B	14	0.6	A/I	B	N	N
				LE	6/60 NIP	528	16	0.7	A/S/I		B	125	122	74	98	16	0.7	A/I	B	14	0.7	A/I	B		
46	MARGABANDHU	51	M	RE	6/18 PH 6/12	492	14	0.5	R/PC	HT	B	130	114	74	128	14	0.5	R/PC	B	14	0.5	R/PC	B	N	N
				LE	6/18 PH 6/6	497	14	0.7	R/S/I		B	110	98	65	99	14	0.7	R/S/I	B	14	0.7	R/S/I	B		
47	PREMA	52	F	RE	5/60 PH 6/36	520	16	0.6	R/S/I	-	B	126	139	82	86	14	0.6	R/S	B	14	0.6	R/S	B	N	N
				LE	6/60 PH 6/12	526	16	0.6	R/S		B	123	156	67	96	14	0.6	R/S	B	14	0.6	R /PC	B		
48	JAGATHA	60	F	RE	6/18 PH 6/12	540	16	0.4	R/PC	HT	B	147	135	68	102	14	0.6	A/S	L	14	0.6	A/S	L	N	N
				LE	6/12 PH 6/6	546	16	0.6	R/S/I		B	134	124	76	98	14	0.6	R/S	B	14	0.6	R/S	B		
49	PATCHAIAMMAL	44	F	RE	6/36 PH 6/18	530	18	0.6	R/S/I	HC	B	126	112	75	100	16	0.6	R/S	B	16	0.6	R/S	B	N	N
				LE	6/36 PH 6/6	532	18	0.7	R/S/I		B	123	110	78	94	16	0.7	R/S/I	B	16	0.7	R/S	B		
50	HABINISA	65	F	RE	6/60 PH 6/24	546	16	0.5	R/I	)M/HT	B	135	146	104	86	14	0.5	R/I	B	14	0.5	R/I	B	N	N
				LE	6/12 PH 6/6	535	16	0.7	R/PC		B	125	130	74	86	16	0.7	R/PC	B	14	0.7	R/PC	B		
51	THILAGAN	65	M	RE	6/12 PH 6/9	522	14	0.6	A/F	-	B	129	145	68	130	12	0.6	A/F	B	12	0.6	A/F	B	N	N
				LE	6/60 PH 6/18	520	14	0.5	A /S/I		B	130	114	57	125	12	0.5	R/ S/I	B	12	0.5	R/S/I	B		
52	KAVITHA	45	F	RE	6/36 PH 6/6	532	14	0.7	A/S	-	B	122	98	66	54	14	0.7	A/S/I	L	14	0.8	A/S/I/F	TRAE	N	N

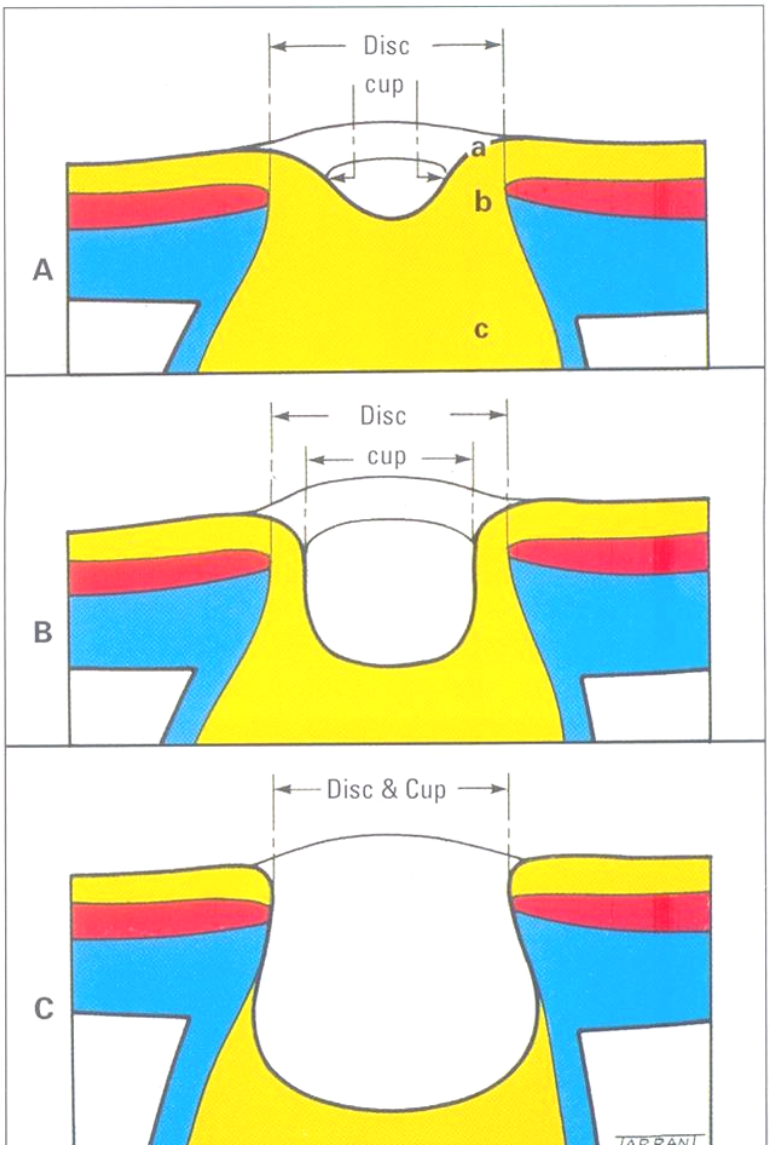
				LE	6/36 PH 6/9	538	14	0.7	A/S/I		B	110	107	86	99	14	0.7	A/ S I	B	14	0.7	A/ S I	B		
53	BALASUBRAMANIAN	65	M	RE	6/24 PH 6/9	460	15	0.7	A/ I	HT	B	124	108	75	87	14	0.7	A /I	B	14	0.7	A / I	B	N	ABN
				LE	6/12 PH 6/6	471	13	0.7	A/ PC/F		B	120	106	78	90	12	0.7	A/ F	B	12	0.7	A/ F	B		
54	SAMPATH	51	M	RE	6/24 PH 6/6	540	12	0.6	A/ F	-	B	130	120	78	99	12	0.6	A/ F	B	12	0.6	A/F	B	N	N
				LE	6/36 PH 6/6	543	14	0.4	A / I		B	140	147	74	125	14	0.4	A/ I	B	14	0.4	A/I	B		
55	MADHAVAN	55	M	RE	6/24 PH 6/6	540	16	0.6	A/ I /F	-	B	125	112	78	102	14	0.6	A/F	B	14	0.6	A/F	B	N	N
				LE	6/24 PH 6/6	542	16	0.7	R/ S		B	124	116	76	90	14	0.7	R/PC	B	14	0.7	R/PC	B		
56	MAHADEVAN	53	M	RE	6/9 PH 6/6	539	14	0.8	A/S/I	HM	B	118	108	68	67	14	0.8	A/S/I	B	14	0.8	A/S/I	B	DEC	N
				LE	6/9 PH 6/6	530	18	0.7	A/S		B	121	110	72	75	16	0.7	A/S	B	16	0.7	A/S	B		
57	RUDRAVENI	45	F	RE	6/12 NIP	528	16	0.8	A/S/F	-	B	135	107	78	65	16	0.8	A/S/F	B	14	0.8	A/S/F	B	N	N
				LE	6/12 PH 6/6	521	14	0.5	R/S		B	133	134	76	121	14	0.5	R/S	B	14	0.5	R/S	B		
58	HAMSAKUMARI	50	F	RE	6/24 PH 6/9	530	14	0.7	A/S/F	HT	B	122	110	78	95	12	0.7	A/S/F	B	14	0.7	A/S	B	N	N
				LE	6/12 PH 6/6	540	14	0.7	A/ S		B	126	128	82	89	12	0.7	A/S	B	14	0.7	A/S	B		
59	VEDHANAYAGAM	55	M	RE	6/24 PH 6/6	486	14	0.6	A/ S/F	HT	B	136	130	98	102	14	0.6	A/S	B	14	0.6	A/S	B	N	N
				LE	6/12 PH 6/9	483	14	0.6	R/S/I		B	143	134	95	100	12	0.6	R/S	B	12	0.6	R/S	B		
60	VELU	60	M	RE	6/12 PH 6/6	530	14	0.7	A/F	DM	B	126	100	56	91	14	0.7	A/ F	B	12	0.7	A/F	B	N	N
				LE	6/36 PH 6/12	540	12	0.5	R/PC		B	123	101	83	96	12	0.5	R/PC	B	12	0.5	R/PC	B		
61	KAANTHA	55	F	RE	6/24 PH 6/9	538	12	0.6	R/PC/F	-	B	124	155	65	114	12	0.6	R/PC	B	12	0.6	R/PC	B	N	N
				LE	6/12 PH 6/6	530	16	0.8	A/S/I		B	120	110	55	98	16	0.8	A/S/I	B	14	0.8	A/S/I	B		
62	PUSHPALATHA	55	F	RE	6/24 PH 6/6	534	16	0.7	A/S/F	DM	B	126	112	66	90	14	0.7	A/S/F	B	14	0.7	A/S/F	B	N	N
				LE	6/24 PH 6/9	540	14	0.6	R/S		B	122	108	82	93	14	0.6	R/PC	B	14	0.6	R/PC	B		
63	KUPPAMAL	40	F	RE	6/12 PH 6/6	535	14	0.5	R/S	-	B	135	120	79	77	14	0.5	R/F	B	14	0.5	R/F	B	N	N
				LE	6/24 PH 6/6	537	14	0.5	R/F		B	136	130	65	110	14	0.5	R/PC	B	14	0.5	R/PC	B		
64	KARTHIKEYAN	55	M	RE	6/18 PH 6/6	547	16	0.6	A/I	-	B	124	155	67	115	14	0.6	A/I	B	14	0.6	A/I	B	N	N
				LE	6/18 PH 6/9	542	14	0.4	R/S		B	153	139	71	108	14	0.4	R/S	B	14	0.4	R/S	B		
65	SWARNALATHA	70	F	RE	6/36 PH 6/9	530	16	0.5	R/PC	-	B	133	130	78	120	14	0.5	R/PC	B	14	0.5	R/PC	B	N	N
				LE	6/24 PH 6/9	527	16	0.6	R/S/I		B	140	156	77	127	14	0.6	R/S/I	B	14	0.6	R/S	B		
66	ELUMALAI	60	M	RE	6/36 PH 6/6	581	15	0.7	A/S/I	-	B	141	138	84	101	12	0.7	A/S/I	B	14	0.7	A/S/I	B	N	N
				LE	6/60 PH 6/9	583	15	0.7	A/I		B	161	154	91	96	12	0.7	A/I	B	14	0.7	A/I	B		
67	SAMPATH	65	M	RE	6/60 PH 6/24	530	16	0.6	A/PC/F	DM	B	134	107	77	88	14	0.6	A/PC/F	B	14	0.6	A/PC/F	B	N	N
				LE	6/24 PH 6/12	522	16	0.7	A/S		B	103	92	78	89	14	0.7	A/S	B	14	0.7	A/S	B		
68	VARADHARAJAN	55	M	RE	6/12 PH 6/6	512	14	0.5	R/S/I	HOT	B	131	122	69	106	12	0.5	R/S	B	12	0.5	R/S	B	N	N
				LE	6/18 PH 6/9	517	14	0.7	A/S/I		B	93	102	79	97	14	0.7	A/S/I	B	14	0.7	A/S/I	B		
69	KANNAN	48	M	RE	6/12 PH 6/6	539	14	0.7	A/S/I	-	B	116	106	86	98	12	0.7	A/S	B	14	0.7	A/S	B	N	N
				LE	6/12 PH 6/6	536	14	0.5	R/S/F		B	138	154	100	102	12	0.5	R/S/F	B	14	0.5	R/S	B		

70	RAVIKUMAR	43	M	RE	6/24 PH 6/6	540	14	0.6	R/S/I	ABL	B	134	107	88	77	14	0.6	R/S	B	14	0.6	R/S	B	N	N
				LE	6/12 PH 6/6	543	14	0.5	R/S		B	107	153	84	112	14	0.5	R/S	B	14	0.5	R/S	B		
71	VANITHA	35	F	RE	6/36 PH 6/9	562	16	0.7	A/S/F	-	B	122	135	86	102	14	0.7	A/S/F	B	14	0.7	R/S	B	N	N
				LE	6/24 PH 6/9	559	14	0.6	A/F		B	124	132	96	94	14	0.6	A/F	B	14	0.6	A/F	B		
72	PARAMESWARAN	50	M	RE	6/9 PH 6/6	527	14	0.5	R/S	IHD	B	119	126	78	91	14	0.5	R/PC	B	14	0.5	R/PC	B	N	N
				LE	6/9 PH 6/6	523	14	0.6	R/S/I		B	121	130	49	105	14	0.6	R/S/I	B	14	0.6	R/S/I	B		
73	SASIKALA	45	F	RE	6/24 PH 6/6	540	16	0.7	A/S/I	-	B	125	152	63	119	16	0.7	A/S/I	B	14	0.7	A/S/I	B	N	N
				LE	6/18 PH 6/6	545	16	0.6	A/S/F		B	150	154	84	79	14	0.6	A/S/F	B	14	0.6	A/S/F	B		
74	MEENAKSHI	67	F	RE	6/60 PH 6/9	522	16	0.6	A/F	-	B	109	143	100	120	16	0.6	A/F	B	16	0.6	A/F	B	N	N
				LE	6/36 PHH 6/6	526	14	0.5	R/PC		B	156	151	87	96	16	0.5	R/PC	B	14	0.5	R/PC	B		
75	SUBASH	60	M	RE	6/18 PH 6/12	540	16	0.6	A/I	DM	B	149	111	80	106	16	0.6	A/I	B	14	0.6	A/I	B	N	N
				LE	6/60 NIP	543	16	0.7	A/S/I		B	108	86	68	129	16	0.7	A/I	B	14	0.7	A/I	B		
76	SARASWATHI	52	F	RE	6/18 PH 6/12	536	14	0.5	R/PC	-	B	130	142	93	119	14	0.5	R/PC	B	14	0.5	R/PC	B	N	N
				LE	6/18 PH 6/6	538	14	0.7	R/S/I		B	110	126	88	100	14	0.7	R/S/I	B	14	0.7	R/S/I	B		
77	ARUMUGAM	45	M	RE	5/60 PH 6/36	542	16	0.6	R/S/I	-	B	112	102	90	95	14	0.6	R/S	B	14	0.6	R/S	B	N	N
				LE	6/60 PH 6/12	547	16	0.6	R/S		B	102	94	88	96	14	0.6	R/S	B	14	0.6	R /PC	B		
78	VENUGOPAL	62	M	RE	6/18 PH 6/12	540	16	0.4	R/PC	-	B	105	140	75	109	14	0.4	R/PC	B	14	0.4	-	B	N	N
				LE	6/12 PH 6/6	546	16	0.6	R/S/I		B	120	118	68	95	14	0.6	R/S	B	14	0.6	R/S	B		
79	MANOHARAN	50	M	RE	6/36 PH 6/18	490	18	0.6	R/S/I	DM	B	119	104	72	97	16	0.6	R/S	B	16	0.6	R/S	B	N	N
				LE	6/36 PH 6/6	489	18	0.7	R/S/I		B	108	102	68	90	16	0.7	R/S/I	B	16	0.7	R/S	B		
80	RAMASAMY	65	M	RE	6/60 PH 6/24	546	16	0.5	R/I	-	B	103	130	75	100	14	0.5	R/I	B	14	0.5	R/I	B	N	N
				LE	6/12 PH 6/6	535	16	0.7	A/S		B	141	113	90	67	16	0.7	A/S	B	14	0.7	A/S	B		
81	JAYA	60	F	RE	6/12 PH 6/9	522	15	0.6	A/F	-	B	109	126	88	94	12	0.6	A/F	B	12	0.6	A/F	B	N	N
				LE	6/60 PH 6/18	553	14	0.5	A /S/I		B	125	118	93	95	12	0.5	R/ S/I	B	12	0.5	R/S/I	B		
82	SUNDARAMURTHY	55	M	RE	6/36 PH 6/6/	528	14	0.7	R/PC	HC	B	124	132	62	96	14	0.7	R/ S/I	L	14	0.7	R/S/I	L	N	ABN
				LE	6/36 PH 6/9	530	14	0.7	A / S I		B	122	102	74	86	14	0.7	A/ S I	B	14	0.7	A/ S I	B		
83	KARNAN	48	M	RE	6/24 PH 6/6	535	14	0.7	A/S/I	-	B	126	100	75	96	14	0.7	A/S/I	B	14	0.7	A/S/I	B	N	N
				LE	6/36 PH 6/9	545	15	0.6	A/F		B	102	130	72	105	12	0.6	A/F	B	14	0.6	A/F	B		
84	SAMPATH KUMAR	70	M	RE	6/24 PH 6/18	550	16	0.7	R/S/I	IHD	B	135	114	63	102	14	0.7	R/S/I	B	14	0.7	R/S	B	N	N
				LE	6/18 PH 6/6	548	16	0.5	R/S		B	114	131	99	73	14	0.5	R/S	B	14	0.5	R/S	B		
85	KONDIAMAL	53	M	RE	6/12 PH 6/6	550	14	0.6	A/S	-	B	128	96	93	72	14	0.6	A/S	B	14	0.6	R/S	B	N	N
				LE	6/18 PH 6/6	552	14	0.7	A /S /I		B	135	114	62	104	12	0.7	A/S/I	B	14	0.7	A/S/I	B		
86	BAKIAVATHY	55	M	RE	6/24 PH 6/6	529	14	0.7	R/PC	HC	B	124	154	71	105	14	0.7	A/S	L	14	0.7	R/S/I	L	N	ABN
				LE	6/24 PH 6/6	520	15	0.7	A/S/I		B	129	120	62	105	12	0.7	A/ S I	B	14	0.7	A/ S I	B		
87	RAMALINGAM	65	M	RE	6/9 PH 6/6	530	14	0.7	A/ I	HT	B	133	104	59	102	14	0.7	A /I	B	14	0.7	A / I	B	N	N

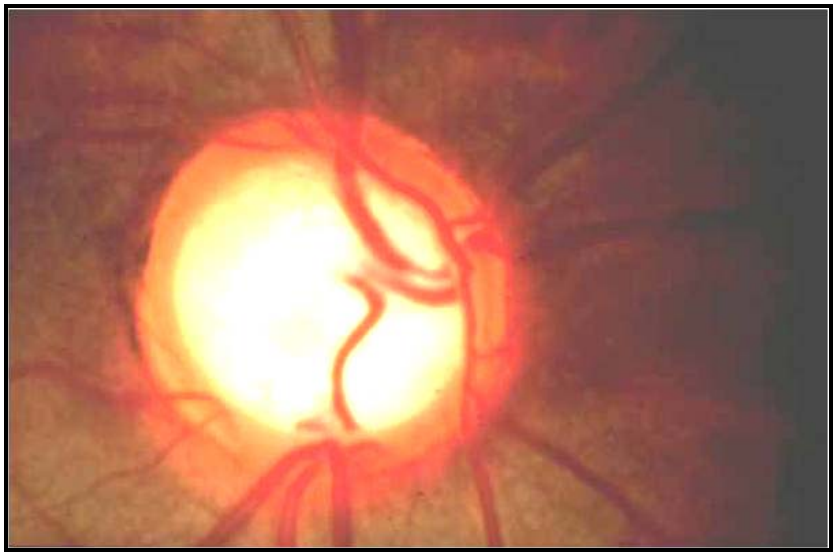
				LE	6/9 PH 6/6	527	12	0.7	A/ PC/F		B	110	96	60	100	12	0.7	A/ F	B	12	0.7	A/ F	B		
88	DEVARAJ	50	M	RE	6/12 NIP	540	12	0.6	A/ F	-	B	142	142	89	100	12	0.6	A/ F	B	12	0.6	A/F	B	N	N
				LE	6/12 PH 6/6	543	14	0.4	A / I		B	139	143	78	89	12	0.4	A/ I	B	14	0.4	A/I	B		
89	MOHANKUMAR	54	M	RE	6/24 PH 6/9	530	16	0.6	A/ I /F	-	B	155	142	75	130	14	0.6	A/F	B	14	0.6	A/F	B	N	N
				LE	6/12 PH 6/6	528	16	0.7	R/ S		B	126	130	70	95	14	0.7	R/PC	B	14	0.7	R/PC	B		
90	MARAGATHAM	58	F	RE	6/60 NIP	529	14	0.8	A/S/I	HM	B	124	126	74	90	12	0.8	A/S/I	B	14	0.8	A/S/I	B	DEC	N
				LE	6/12 PH 6/9	530	18	0.7	A/S		B	136	130	80	94	16	0.7	A/S	B	16	0.7	A/S	B		
91	PUSHPARAJ	60	M	RE	6/12 PH 6/6	532	16	0.8	A/S/F	-	B	132	125	75	90	16	0.8	A/S/I	L	16	0.8	A/S/I/F	TRAE	N	N
				LE	6/60 NIP	536	14	0.5	R/S		B	110	112	86	76	14	0.5	R/S	B	14	0.5	R/S/F	B		
92	JANAKI	58	F	RE	6/24 PH 6/9	521	15	0.7	A/S/F	HT	B	121	123	68	98	14	0.7	A/S/F	B	14	0.7	A/S	B	N	N
				LE	6/36 NIP	530	14	0.7	A/ S		B	122	120	74	91	14	0.7	A/S	B	14	0.7	A/S	B		
93	RAJALAKSHMI	55	F	RE	6/24 PH 6/6	536	14	0.6	A/ S/F	HT	B	100	128	82	105	14	0.6	A/S	B	14	0.6	A/S	B	N	N
				LE	6/24 PH 6/9	535	14	0.6	R/S/I		B	125	120	65	104	12	0.6	R/S	B	12	0.6	R/S	B		
94	JACQUILINE	45	F	RE	6/12 PH 6/6	530	15	0.7	A/F	DM	B	126	127	71	77	14	0.7	A/ F	B	12	0.7	A/F	B	N	N
				LE	6/24 PH 6/6	540	12	0.5	R/PC		B	117	116	54	76	12	0.5	R/PC	B	12	0.5	R/PC	B		
95	RAMESHKUMAR	53	M	RE	6/18 PH 6/6	528	12	0.6	R/PC/F	-	B	127	95	93	73	12	0.6	R/PC	B	12	0.6	R/PC	B	N	N
				LE	6/24 PH 6/9	530	16	0.8	A/S/I		B	122	104	62	70	16	0.8	A/S/I	B	14	0.8	A/S/I	B		
96	HEMALATHA	35	F	RE	6/36 PH 6/12	534	16	0.7	A/S/F	DM	B	135	114	62	102	14	0.7	A/S/F	B	14	0.7	A/S/F	B	N	N
				LE	6/24 PH 6/9	540	14	0.6	R/S		B	128	96	93	72	14	0.6	R/PC	B	14	0.6	R/PC	B		
97	SUBRAMANI	50	M	RE	6/24 NIP	535	14	0.5	R/S	HT	B	128	120	44	84	12	0.5	R/F	B	14	0.5	R/F	B	N	N
				LE	6/24 PH 6/9	540	12	0.5	R/PC	-	B	138	119	55	88	12	0.5	R/PC	B	12	0.5	R/PC	B	N	N
98	GEETHALAKSHMI	58	M	RE	6/60 PH 6/12	490	12	0.6	R/PC/F		B	110	140	56	109	12	0.6	R/PC	B	12	0.6	R/PC	B		
				LE	6/24 PH 6/6	495	16	0.8	A/S/I	-	B	125	150	63	47	16	0.8	A/S/I	B	14	0.8	A/S/I	B	N	N
99	KUMARAN	60	M	RE	6/60 PH 6/36	534	16	0.7	A/S/F	ABL	B	150	154	84	79	14	0.7	A/S/F	B	14	0.7	A/S/F	B		
				LE	6/60 PH 6/18	540	14	0.6	R/S	-	B	131	171	78	100	14	0.6	R/PC	B	14	0.6	R/PC	B	N	N
100	SEETHA	62	F	RE	6/36 PH 6/12	520	14	0.5	R/S		B	125	118	96	93	14	0.5	R/F	B	14	0.5	R/F	B		
				LE	6/24 PH 6/6	527	14	0.5	R/F	-	B	102	130	72	105	14	0.5	R/PC	B	14	0.5	R/PC	B	N	N

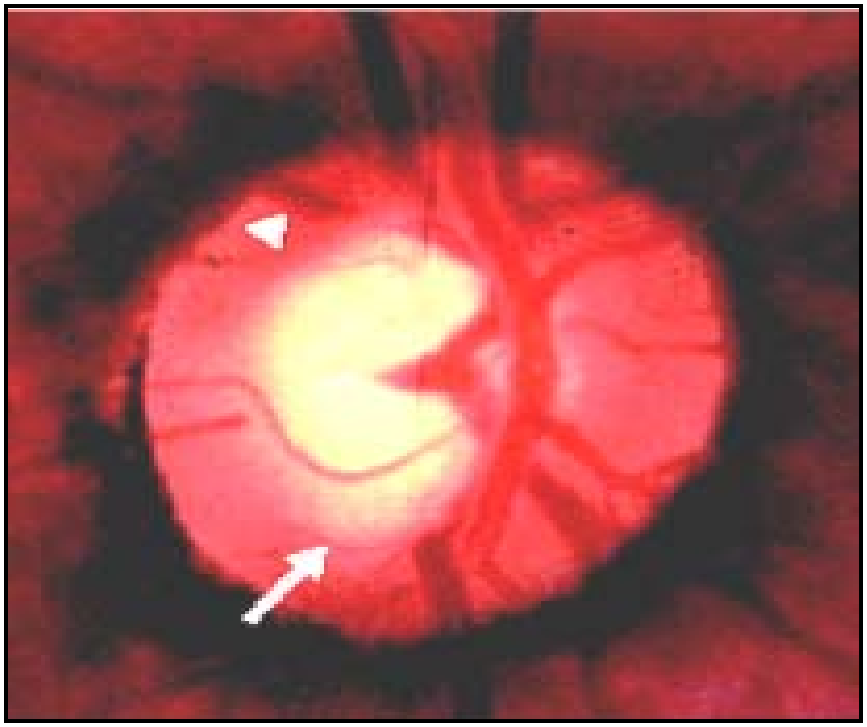
PATHOGENESIS OF OPTIC NERVE HEAD CHANGES IN GLAUCOMA



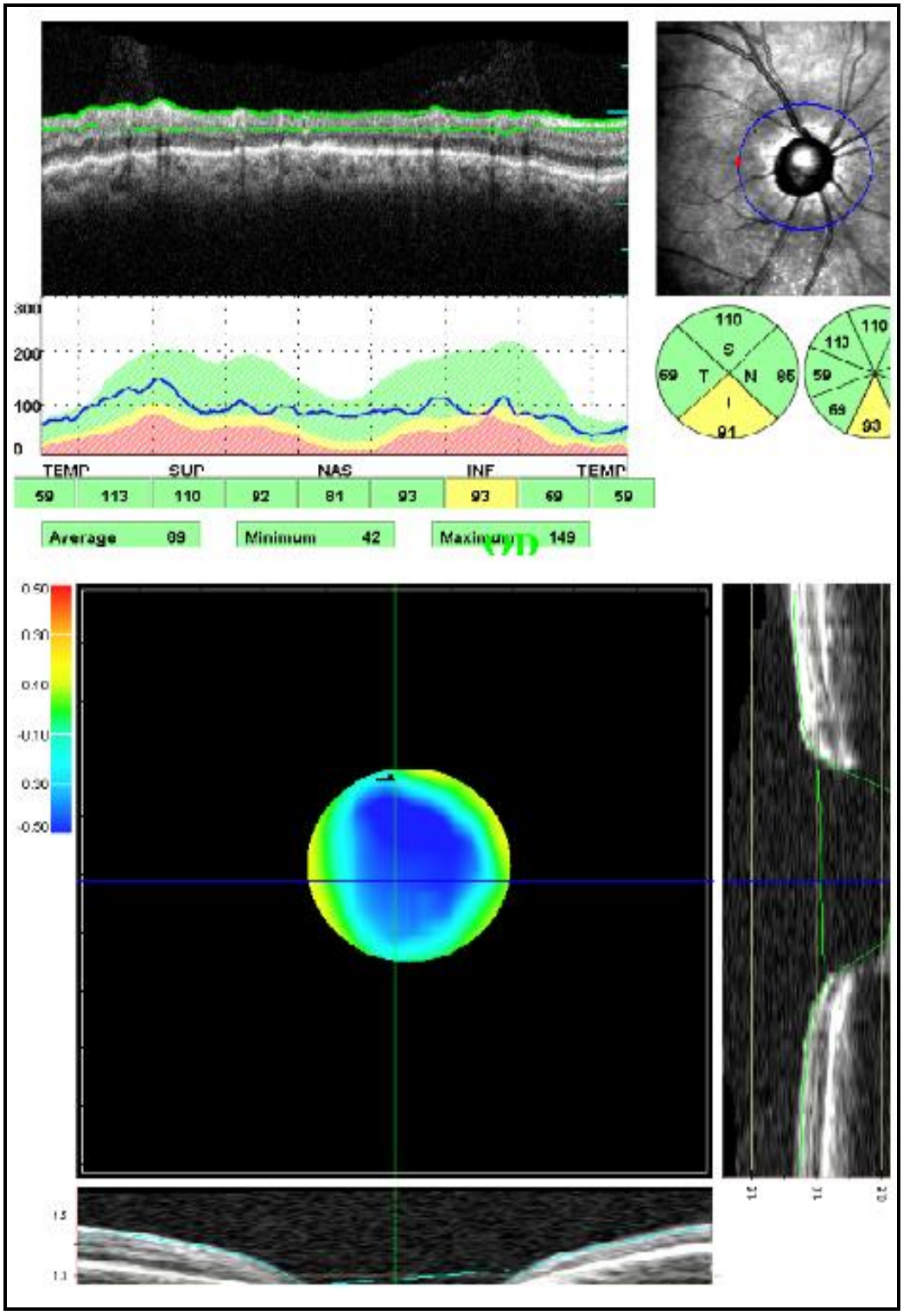


FUNDUS CHANGES IN GLAUCOMA



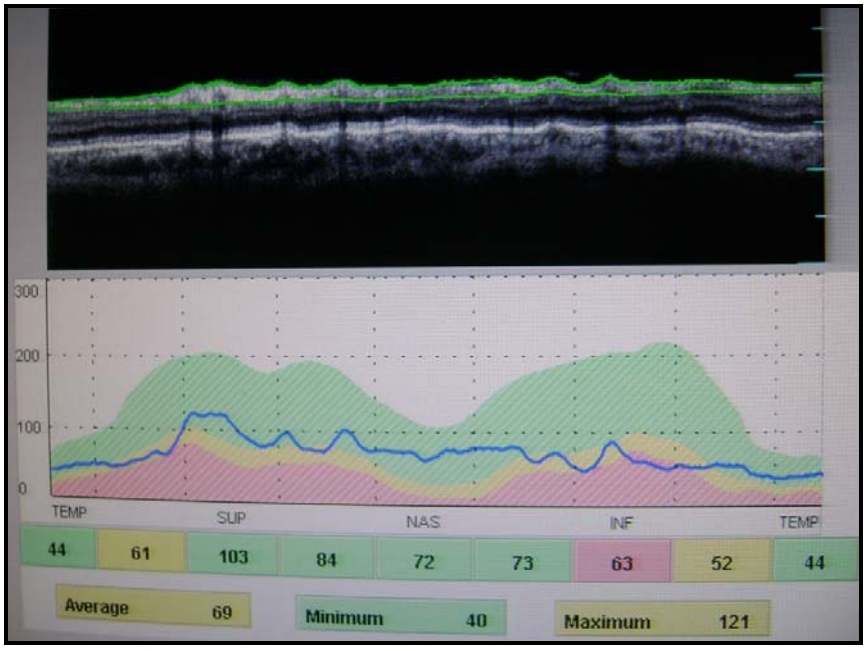


OPTICAL COHERENCE TOMOGRAPHY

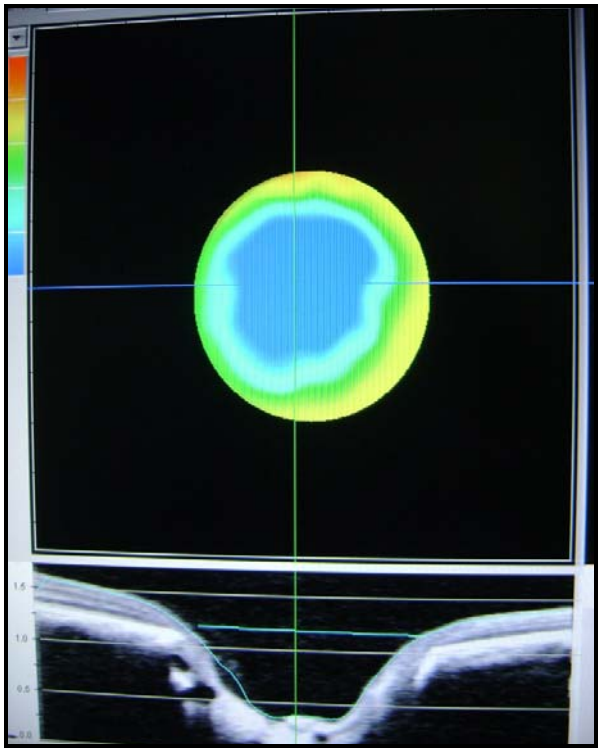


OPTICAL COHERENCE TOMOGRAPHY

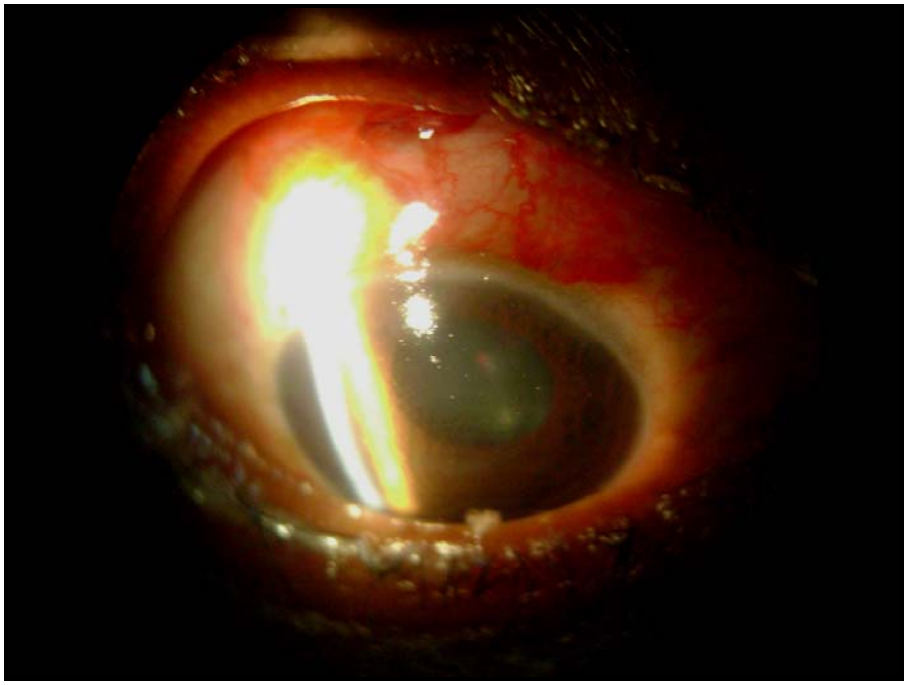
RETINAL NERVE FIBRE LAYER THICKNESS MAP

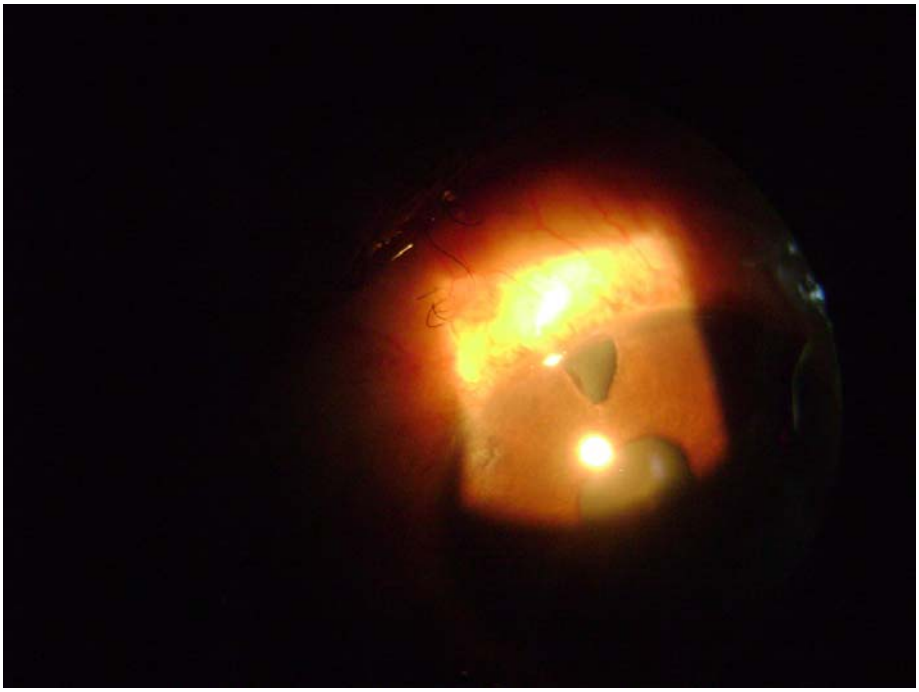


OPTIC NERVE HEAD TOPOGRAPHY

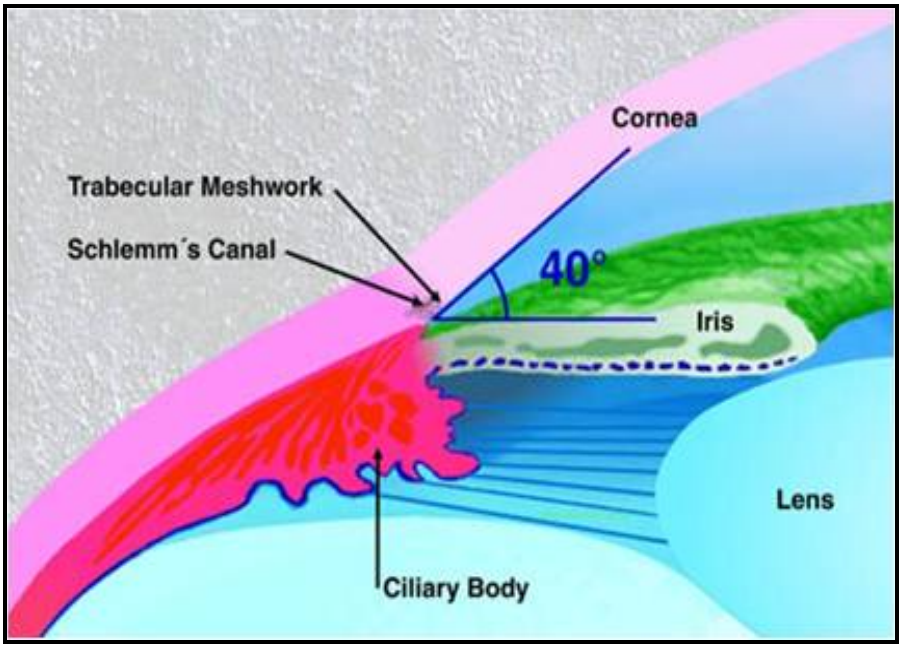


POST OPERATIVE PICTURE SHOWING FILTERING BLEB

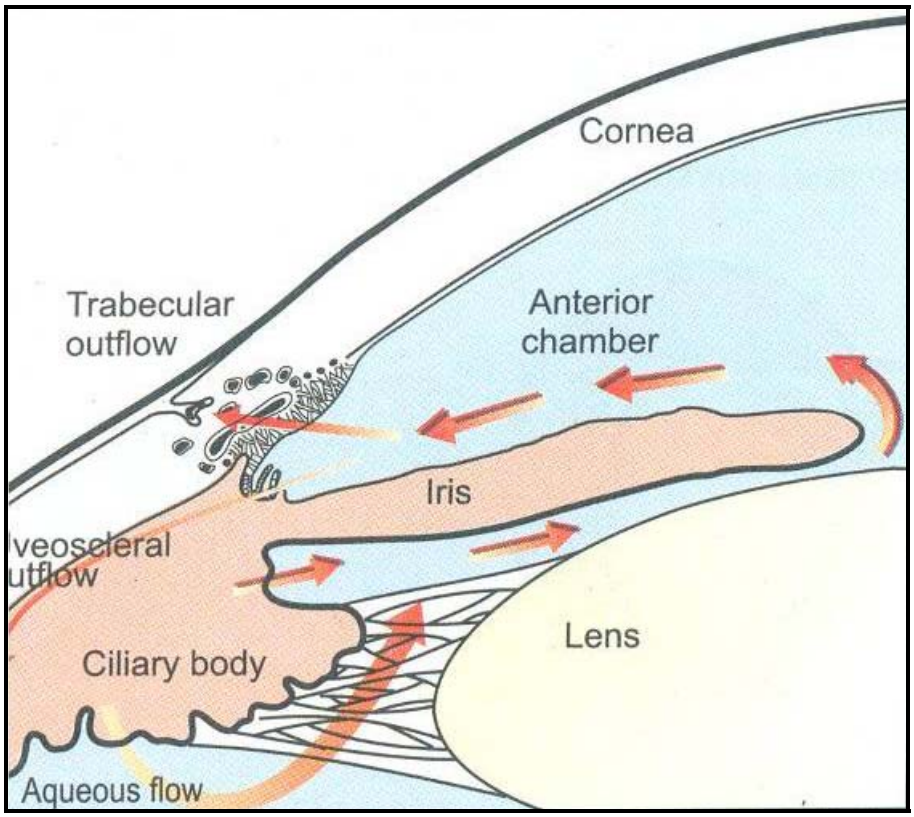




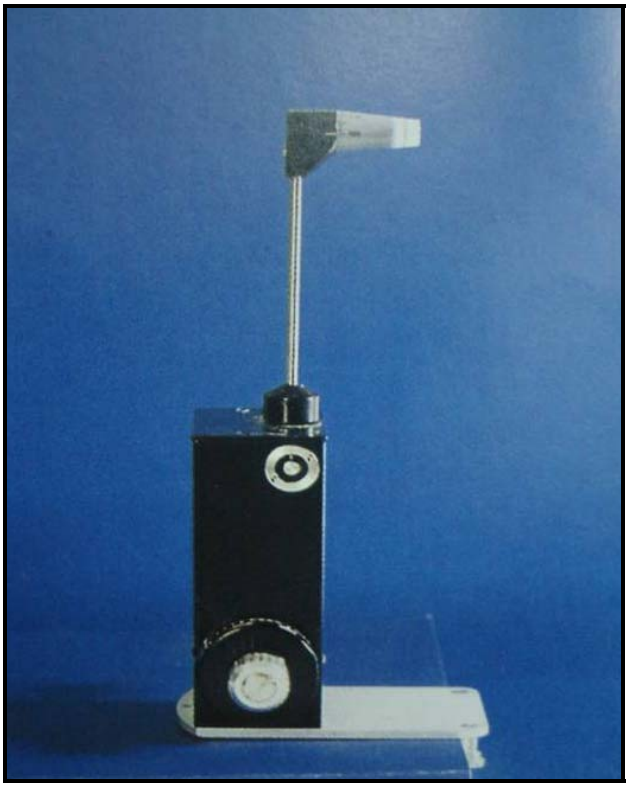
ANATOMY OF TRABECULAR MESHWORK

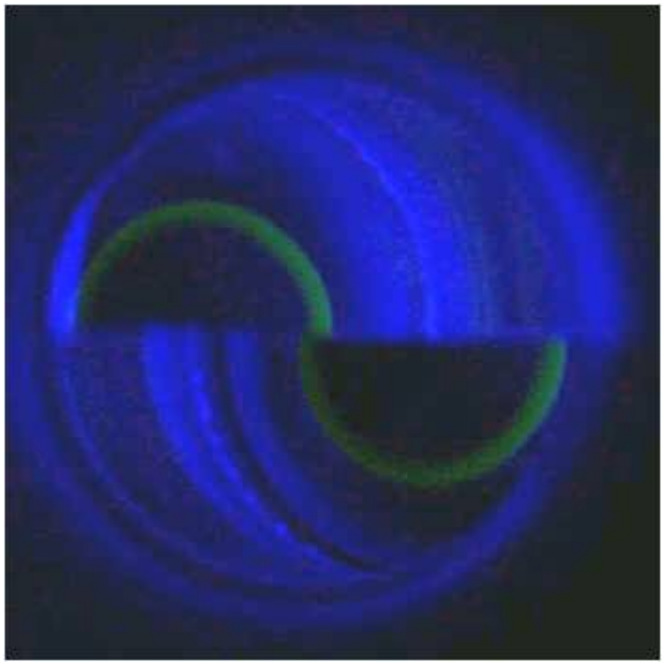


AQUEOUS OUTFLOW PATHWAY

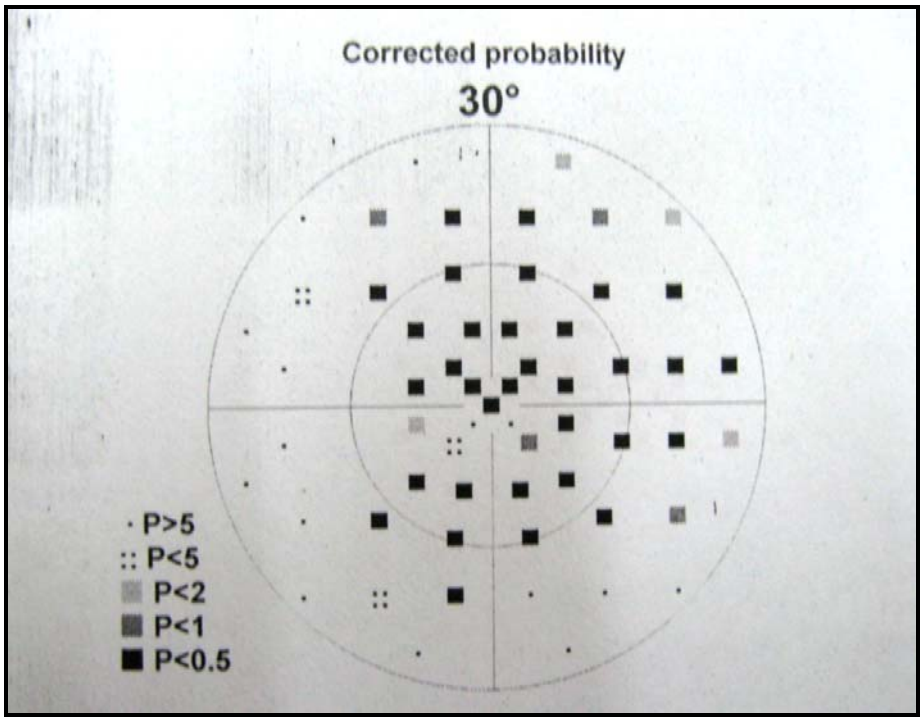


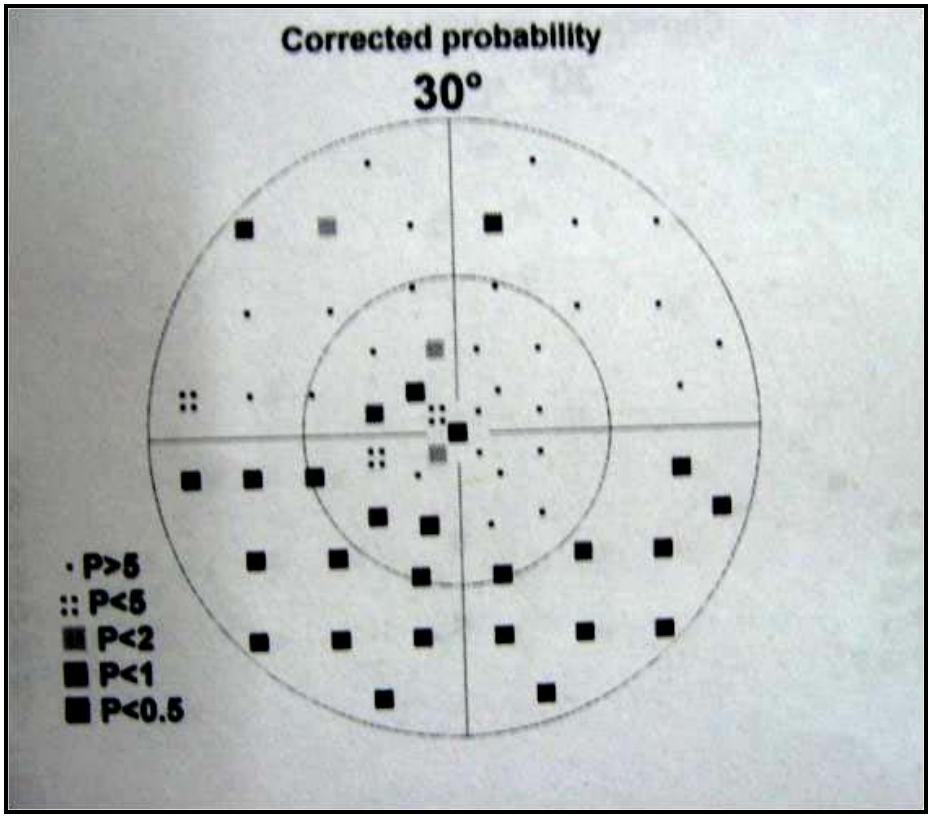
GOLDMANN APPLANATION TONOMETRY



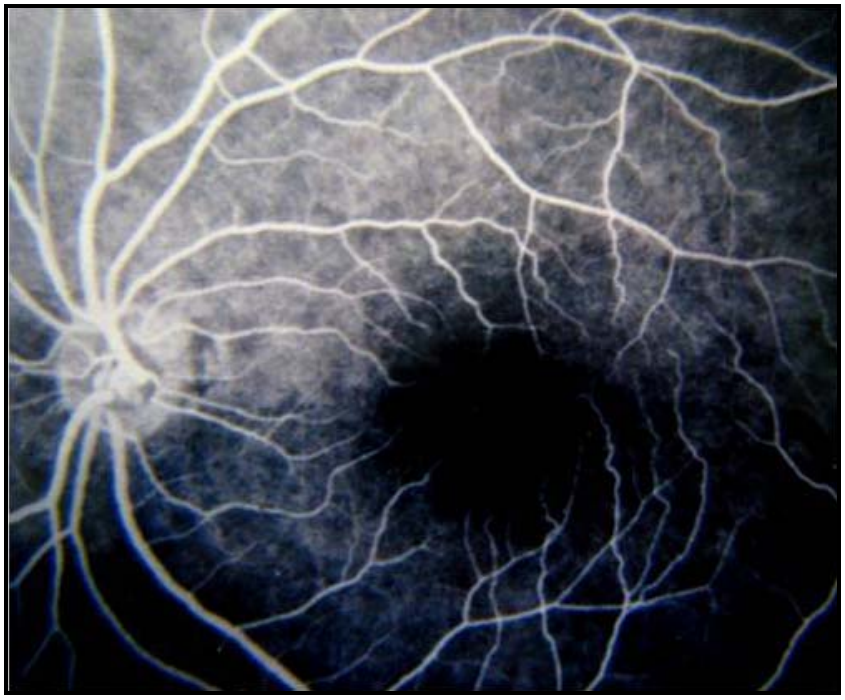


AUTOMATED PERIMETRY REPORT SHOWING DEFECTS INVOLVING FIXATION AREA

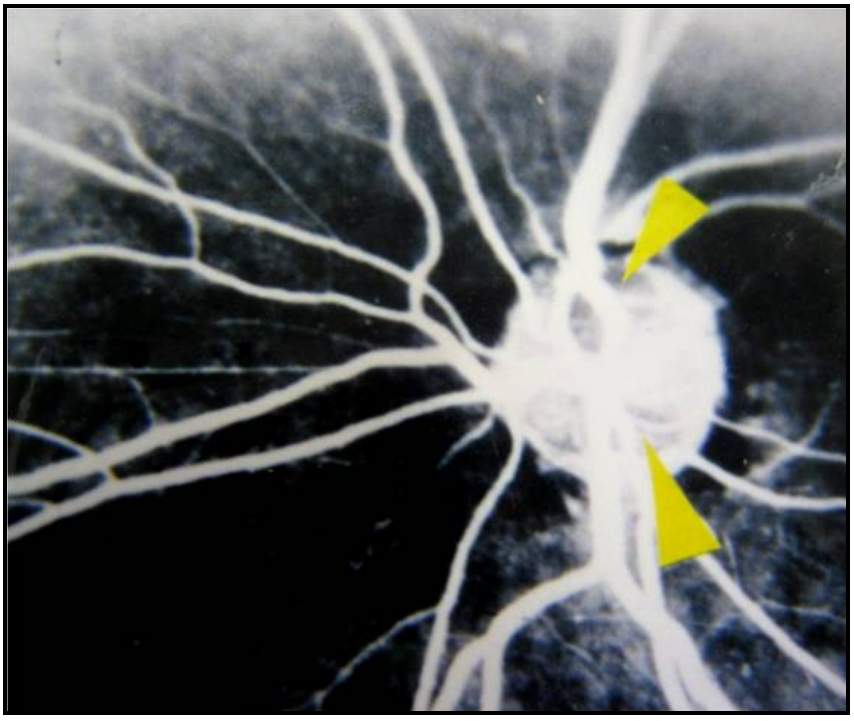




FUNDUS FLUORESCEIN ANGIOGRAPHY OF THE OPTIC NERVE HEAD AND RETINA



FILLING DEFECTS ON THE OPTIC NERVE HEAD



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KEY TO MASTER CHART

NO	-	SERIAL NUMBER
V/A	-	VISUAL ACUITY
PH	-	PIN HOLE
CCT	-	CENTRAL CORNEAL THICKNESS
CIOP	-	CCT CORRECTED INTRAOCULAR PRESSURE
C:D	-	CUP: DISC RATIO
AP	-	FIELDS BY AUTOMATED PERIMETRY
A	-	ABSOLUTE DEFECTS
R	-	RELATIVE DEFECTS
S	-	SUPERIOR ARCUATE AREA
I	-	INFERIOR ARCUATE AREA
F	-	FIXATION AREA
PC	-	PARACENTRAL AREA
RF	-	RISK FACTORS
HT	-	HYPERTENSION
HOT	-	HYPOTENSION
DM	-	DIABETES MELLITUS
IHD	-	ISCHEMIC HEART DISEASE
HC	-	HYPERCHOLESTEROLEMIA
HM	-	HEMORRHOIDS
ABL	-	ACUTE BLOOD LOSS
MG	-	MIGRAINE
PVD	-	PERIPHERAL VASCULAR DISEASE
TRT	-	TREATMENT

RNFL	-	RETINAL NERVE FIBER LAYER THICKNESS
SUP	-	SUPERIOR
INF	-	INFERIOR
TEM	-	TEMPORAL
NAS	-	NASAL
B	-	BRIMONIDINE EYE DROPS TWICE DAILY
L	-	LATANOPROST EYE DROPS ONCE DAILY AT BED TIME
BL	-	BETAXOLOL EYE DROPS TWICE DAILY
TRA	-	TRABECULECTOMY
P1	-	IOP ON FOLLOW UP 1
P2	-	IOP ON FOLLOW UP 2
HB %	-	HEMOGLOBIN
LP	-	LIPID PROFILE
N	-	NORMAL
ABN	-	ABNORMAL
DEC	-	DECREASED

PROFORMA FOR THE CLINICAL ANALYSIS OF NORMAL TENSION GLAUCOMA

NAME: ADDRESS:

AGE: SEX:

G.C.No: PHONE NO:

HISTORY:

- DEFECTIVE VISION :RE/LE/BE DURATION:
- HEADACHE
- FREQUENT CHANGE OF SPECTACLES
- TRAUMA
- RECURRENT ATTACKS OF REDNESS / PAIN/ WATERING
- H/O STEROID INTAKE

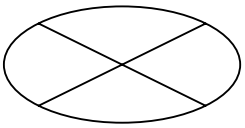
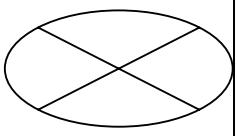
RISK FACTORS:

	TICK IF YES	**DURATION**	**TREATMENT**
DIABETES			
HYPERTENSION			
HYPOTENSION			
ISCHEMIC HEART DISEASE			
HEADACHE/ MIGRAINE			
HYPERCHOLESTEROLEMIA			
HEMORRHOIDS			
PERIPHERAL VASCULAR DISEASE			
ACUTE BLOOD LOSS- RTA			
IMMUNOLOGICAL DISORDER			

PAST HISTORY :

PREVIOUS HISTORY OF INTRAOCULAR SURGERY

OCULAR EXAMINATION:

	RE	**LE**
V/A(BCVA)		
IOP		
CCT		
CCT CORRECTED IOP		
ANTERIOR SEGMENT		
FUNDUS: MEDIA: C:D RATIO: PALLOR: NASALISATION: BAYONETTING: LAMINAR DOT SIGN: SPLINTER HEMORRHAGE: PERIPAPILLARY ATROPHY:		
GONIOSCOPY:		
AUTOMATED PERIMETRY: RELIABILITY: DEFECTS: REGION: FIXATION AREA:		

DIURNAL IOP MONITORING:

TIME	RE	LE

TREATMENT:--

OCT:

RNFL THICKNESS		
SUPERIOR:		
INFERIOR:		
TEMPORAL:		
NASAL:		

LAB INVESTIGATIONS:

1. HEMOGLOBIN:
2. LIPID PROFILE:

FFA: ARM-CHOROIDAL FILLING TIME:

ARTERIO-VENOUS TRANSIT PHASE:

FILLING DEFECTS ON THE ONH:

FOLLOW UP:

1. DATE:
2. DRUGS:
3. COMPLIANCE:
4. V/A:
5. IOP:
6. ANTERIOR SEGMENT:
7. FUNDUS:
8. AUTOMATED PERIMETRY:
9. STATIC/PROGRESSION/REGRESSION:
10. ADVICE:

LIST OF SURGERIES PERFORMED

S. No	NAME	AGE / SEX	IP. no.	DIAGNOSIS	DATE OF SURGERY	SURGERY PERFORMED
1.	SAROJA	55/F	416107	LE-MC	25.7.07	LE-ECCE WITH PCIOL
2.	RAJI	60/F	418191	BE-IMC	19.9.07	RE-ECCE WITH PCIOL
3.	ELUMALAI	60/M	418270	BE-IMC	20.3.08	LE-ECCE WITH PCIOL
4.	RAJESHWARI	60/F	426798	BE-IMC	30.5.08	LE-SICS WITH PCIOL
5.	RANI	62/F	426021	BE-IMC	6.6.08	RE-SICS WITH PCIOL
6.	KUPPU	70/F	422082	BE-IMC	3.3.09	RE-SICS WITH PCIOL
7.	SUSEELA	62/F	461141	RE-IMC	14.4.09	RE-SICS WITH PCIOL
8.	JOSEPH	60/M	431327	BE-IMC	27.5.08	LE-SICS WITH PCIOL
9.	KALAVATHY	45/F	4222021	RE-NVG	19.8.09	RE-TRABECULECTOMY
10	PONNAMMA	50/F	4322451	RE-CHR.DAC	18.9.09	RE-DCR
11	MALATHY	45/F	4344273	LE-CHR.DAC	9.10.09	LE-DCR
12	PUSHPA	43/F	4314523	RE-PTERYG.	17.6.09	RE-PTERYG. EXCISION WITH AMNIOTIC MEMBRANE

S. No	NAME	AGE / SEX	IP. no.	DIAGNOSIS	DATE OF SURGERY	SURGERY PERFORMED
						GRAFT
13	MOHAMED	46/M	4423122	LE-PTERYG.	15.7.09	LE-PTERYG.EXCISION WITH CONJ.AUTOGRAFT
14	MANJULA	48/F	4432761	RE-PTERYG.	5.8.09	RE-PTERYG.EXCISION WITH AMNIOTIC MEMBRANE GRAFT
15	MALLIKA	50/F	432156	RE-IMC	10.8.09	RE-SICS WITH PCIOL
16	SRINIVASAN	60/M	432257	RE-PSC	24.8.09	RE-SICS WITH PCIOL
17	DHANAPAL	56/M	421343	RE-ENTROPION	18.9.09	RE-LATERAL TARSAL STRIP PROCEDURE
18	KUPAYEE	68/F	427833	RE-CHR.DAC	25.9.09	RE-DACRYOCYSTECTOMY
19	KUMARESAN	65/M	423564	LE-CORNEAL TEAR	16.10.09	LE- CORNEAL TEAR SUTURING DONE
20	MURUGAVEL	50/M	423534	RE-IMC	14.10.09	RE-SICS WITH PCIOL
21	KANDASA	53/M	423345	LE-IMC	21.10.09	LE-SICS WITH

S. No	NAME	AGE / SEX	IP. no.	DIAGNOSIS	DATE OF SURGERY	SURGERY PERFORMED
	MY					PCIOL
22	VELAYAN	50/M	423546	RE-PSC	28.10.09	RE-SICS WITH PCIOL
23	SUBAIYAN	58/M	426754	RE-MC	2.11.09	RE-SICS WITH PCIOL
24	KUPPUSAMY	60/M	426765	LE-PSC	9.11.09	LE-SICS WITH PCIOL
25	MUKILAN	50/M	425656	RE-IMC	25.11.09	RE-SICS WITH PCIOL

ABBREVIATIONS

1. **RE/LE - RIGHT EYE / LEFT EYE**
2. **IMC - IMMATURE CATARACT**
3. **MC - MATURE CATARACT**
4. **PSC - POSTERIOR SUBCAPSULAR CATARACT**
5. **CHR.DAC - CHRONIC DACRYOCYSTITIS**
6. **PTERYG - PTERYGIUM**
7. **ECCE - EXTRACAPSULAR CATARACT EXTRACTION**
8. **SICS – SMALL INCISION CATARACT SURGERY**
9. **PCIOL - POSTERIOR CHAMBER INTRAOCULAR LENS**
10. **DCR - DACRYOCYSTORHINOSTOMY**